

**THE TREATMENT OF DIABETES, WITH SPECIAL REFERENCE  
TO TREATMENT WITH INSULIN AND ITS RESULTS.**

**Submitted for the Degree of**

**Doctor of Medicine**

**of the**

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**by**

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## HISTORY.

When the history of diabetes is reviewed there is revealed the surprising fact that a disease with such characteristic symptoms and complications remained for long unrecognised as a separate entity.

To the ancients it does not appear to have been known. Hippocrates makes no mention of any condition which might be recognised as diabetes. Indeed, the name diabetes ( $\delta\iota\alpha\beta\eta\tau\eta\varsigma$  = a siphon) was not applied to the condition until about 30 A.D. when Aretaeus of Capadocia described a disease in which "the flesh and bones run together into urine", and which presented the characteristic symptoms of polyuria, hunger, thirst, and emaciation.\* So far as is known Galen, 131 A.D. saw two cases of a similar type, and propounded two generalisations on this experience. These were:- (1) that diabetes was a weakness of the kidneys, (2) that the urine consisted of the fluid ingested unchanged. No one appears to have dared to question the truth of these propositions, and subsequent treatment was based upon the assumption that they were correct. That these statements remained unchallenged for about 1500 years says much for the authority of Galen, and provides yet another instance of how the voice of authority may retard the progress of knowledge.

It is not until 500 A.D. that we find a clear description of diabetes. In that year a physician named Aëtius described several cases, and gave in some detail the treatment which he employed. These methods of treatment -

\* "Probably D. Insipidus". 41.

bleeding, emetics, and opium - are of interest as they remained in vogue for hundreds of years despite the changing theories concerning the cause of the disease; and the last, opium, has been the sheet-anchor of all physicians up to the beginning of the present century. It is a curious fact that about the same time there appeared in Hindu medicine (in the Ayur Veda of Susruta) a description of the disease under the name of "honey-urine", so that this sweetness of the urine was known and recorded in India 1100 years before any reference is made to it in English literature.

Ancerima (980 - 1037 A.D.) was the originator of the nervous theory of the origin of diabetes. He considered that the disease was due to a disturbance in the nerve plexus of the kidney, but so strong was the influence of Galen that little attention was paid to his hypothesis. His treatment consisted of repeated emesis and vigorous purgation, a procedure which probably owed its success to its being virtually a "starvation" treatment.

The first Englishman to make a notable contribution to the literature on diabetes was Thomas Willis (1675). Willis observed that the urine was "wonderfully sweet, as if imbued with honey or sugar", and, as the urine was formed from the blood, he considered the disease to be primarily a disease of the blood, which, owing to the absence of salts, was too thin. His treatment, therefore, was a diet of milk, rice, starchy and gummy food, all of which were considered to exert a

thickening influence upon the blood. Frequently he limited patients to a diet of milk and barley water, boiled with small quantities of bread - an undernutrition dietary such as is occasionally prescribed to-day. Salt he gave as lime water; and this, the first alkali to be employed, later came into very general use. Willis advocated, too, a treatment with antimony which had a very considerable vogue for a time; and to him must be ascribed the credit or blame of establishing firmly the popularity of Dover's Powder and other opiates in the treatment of diabetes.

It is of interest to note that Sydenham, one of the greatest of English physicians, has left no valuable contribution to the literature on diabetic treatment. Fruit and vegetables were regarded by him as very unsuitable as articles of diet, while he reported very favourably upon the narcotic treatment as practised by Willis.

The next notable advance was made in 1775 when Matthew Dobson completed the discovery of Willis by proving that the sweetness of the urine was due to sugar. He observed, too, that the blood serum of diabetics tasted sweet, and thus he concluded that the sugar was absorbed from the stomach, there being a defective assimilation of food. An account of Dobson's work was read in 1796 by an Army Surgeon named John Rollo and from this record, together with his own observations on one case, he deduced that diabetes was "a primary and peculiar affection of the stomach". On this theory he proceeded to treat his first case in somewhat original manner; and, as

this method marked the real commencement of the dietetic treatment of the disease and formed the basis of all succeeding dietaries among English physicians, a detailed description of the diet is not without interest. The diet was made up as follows :-

- "Breakfast:       $1\frac{1}{2}$  pints of milk and  $\frac{1}{2}$  pint of lime water mixed together: and bread and butter.
- Lunch:            Plain blood puddings made of blood and suet only.
- Dinner:           Game or old meats which have been long kept, and as far as the stomach may bear; fat and rancid old meats, as pork. To eat in moderation.
- Supper:           The same as breakfast.

The skin to be daily greased with hog's lard, flannel worn next to the skin and an ulceration about the size of half a crown to be maintained opposite each kidney." Ammonium Sulphide was administered several times daily: Wine of Antimony and Tincture of Opium were given at night, "with tobacco and foxglove in reserve as substances diminishing gastric function." On this regime the patient is recorded to have improved, when cabbage, onions, salad, mustard and radishes were added to the diet.

This diet of animal food, as rancid as possible, Rollo believed to prevent the formation of sugar in the stomach, and so removed the stimulus to diuresis which he had noted that sugar possessed. The treatment was everywhere

enthusiastically adopted, and imitators seemed to vie with each other in attempts to make their diets as nauseous as possible. One observer relates of two cases that "both contracted so great a loathing of food that neither of them ate anything for five days, and so got rid of their distempers". Thus, on incorrect reasoning, was evolved what was really a starvation treatment of diabetes; and to us to-day it seems remarkable, in view of such published statements as that given above, that the correct explanation of their success so long eluded all observers. The matter is still more mysterious when we consider the published work of Robert Watt (1808), a Scots doctor, who actually evolved a starvation treatment from the Rollo diet, combining it with bleeding, blistering, antimony powder, and mercury as was customary at that time. He states "As diabetes is so obviously aggravated by too much aliment the first aim of the practitioner should be to remove a portion of that food which, since it does not nourish, must oppress and injure the system". Apparently the value of Watt's observations passed unrecognised, and his work does not appear to have exercised any influence upon subsequent treatment.

As was to be expected Rollo's treatment soon came to be vigorously assailed. The main objection was the very reasonable one that patients could not be induced to adhere to their diets for any length of time. In addition to this there came in time to be recorded many cases which, when

placed suddenly upon this protein-fat dietary, passed rapidly into coma and died. Thus even in England where it had its strongest advocates the Rollo diet fell gradually into disrepute. By 1813 Warren and others strongly recommended a return to a full and unrestricted dietary with large doses of opium.

Despite its ultimate failure the Rollo diet undoubtedly served a useful purpose in directing attention more towards the relative importance of foodstuffs as compared with that of drugs. From this time onward "substitutes" began to make their appearance in the diet of diabetics. The first of these was Bran Bread, introduced by Prout in 1820. This observer regarded diabetes as a form of dyspepsia, with difficulty in dealing with starchy food. In addition to Bran Bread he advocated green vegetables in amount, but he limited both food and drink. As was customary he employed bleeding freely. Prout first drew attention to the frequency of phthisis as a complication in diabetes and stressed the necessity for avoiding chill, and the danger of too reducing a regime.

No advance falls to be recorded in treatment for the next 20 years, but during this time Majendie and Claud Bernard were making tremendous advances in physiological research which had a very important influence upon treatment in later years. In 1838, however, Bouchardat published the results of his researches through which he was led to suspect the pancreas as the casual factor in diabetes from post mortem findings. From practical experience he strongly advocated a return to the



Rollo diet, and he persistently stressed the necessity for strict dieting as the only sound method of treatment. By him the Rollo diet was considerably modified, the rancid fats being replaced by butter and alcohol. Bouchardat allowed only small amounts of food, and he was the first to recognise the value of an occasional fast day to control glycosuria. Green vegetables he employed very extensively. He devised the practice of boiling vegetables twice and throwing away the water in order to reduce their carbohydrate content. To Bouchardat, too, belongs the credit of inventing Gluten Bread, while he was the first to record the improvement in patients which followed moderate exercise. Alkalis - soda bicarbonate, chalk, magnesia, citrates, tartrates, soap, ammonium and potassium salts - he used invariably; and, though he found that the patient was generally benefitted he showed that they exerted no influence upon the glycosuria. Other discoveries of his were Glycerol for use as a sweetening agent, and laevulose and inulin as permissible carbohydrates in diabetes. All these he considered did not cause glycosuria. Bouchardat's regime was carefully tested by the famous physician Trousseau and most enthusiastically supported by him as a great advance on all previous treatment. Trousseau specially advocated all kinds of fruit in addition, and emphasised the value of exercise in reducing glycosuria.

While recognised to-day as a very big step forward in

diabetic treatment Bouchardat's methods were by no means generally accepted even in France. About the same time indeed a French physician named Piorry suggested that, as sugar was lost in diabetes, it would be sound treatment to replace the loss by feeding the patient with sugar. Curiously enough this method of treatment attracted many adherents, among whom was a physiologist named Schiff, a vigorous opponent of Claud Bernard on most problems in biological chemistry. Schiff himself developed diabetes, treated himself with sugar, and died after a brief illness.

Perhaps the best known of nineteenth century workers on diabetes was Pavy, to whom the study of the disease was the one absorbing interest in life. Most of his work was experimental and followed the lines of Claud Bernard, but he differed greatly from that observer in the interpretation of results. He had, however, a very large diabetic practice, but he made practically no useful contribution to treatment beyond a mild condemnation of the use of opium as conferring only a very transitory benefit. As regards diet, he contented himself with reducing the amount of carbohydrate in the food with little further restriction.

It was left to a Roman physician, Cantani, to re-establish the claims of strict dietetic treatment now in danger of being overlooked. Cantani, realising the difficulty

in enforcing dietetic restrictions, refused to undertake a case unless he was permitted to isolate his patient on the lines of the modern Weir-Mitchell treatment. Lean meat and fats alone were permitted at first, with eggs and shell fish in the less severe types. Water with, occasionally, dilute alcohol, was restricted to  $1\frac{1}{2}$  -  $2\frac{1}{2}$  litres per day. As a substitute for carbohydrate he gave lactic acid. Protein was allowed in the form of 500 gms. of cooked<sup>meat</sup>. He encouraged vigorous exercise, for by this means he found that he could diminish and even abolish glycosuria. He insisted upon one starve day per week. His treatment lasted for 3 months at least, six to nine months in obstinate cases. After 2 months of freedom from glycosuria green vegetables were permitted, and later wine, cheese, fruits, nuts and even a little farinaceous food were added. The least trace of glycosuria was followed by 2 months on strict protein-fat diet. During treatment the urine was tested daily: thereafter a weekly test was sufficient. A distinct advance was Cantani's insistence that the urine should be kept sugar-free whenever possible: nor did he consider that any other state of matters was permissible until a thorough trial on the lines given above had been made. Latterly he advocated that weight should always be increased, and preferred a trace of sugar in the urine to undernutrition, points upon which there has been considerable controversy even up to the present day.

In Germany at this time considerable useful but destructive criticism was levelled at existing beliefs by Kulz. By painstaking observation this observer demonstrated the

futility of lactic acid and of glycerol; the inefficiency of Carlsbad and other waters; and the negative effects of soda bicarbonate and arsenic. Bouchardat's work he admired, and he based his own methods of treatment on the regime of that worker. In his methodical way he tested the carbohydrate tolerance of patients for various foodstuffs, and, as the result of this research, he strongly advocated the use of green vegetables in treatment. Kulz also conducted a most elaborate investigation into the effects of exercise upon diabetics. He found that great benefit was derived in strong patients with mild diabetes, but in weak individuals with severe diabetes it was of no service and generally, indeed, harmful. While he did not advocate undernutrition diets or fasting he introduced the testing of carbohydrate tolerance in each individual case, and the calculating of diets according to the calorie requirements. Thus for the first time suitable diets were built up for each case, and a departure was made from the usual general recommendation to "allow" this and to "forbid" that. A large and influential school followed Kulz.

Naunyn, another great name in the history of diabetes, made no noteworthy contribution to the treatment of the disease. In his earlier days he employed the Cantani system of seclusion with rigorously restricted diet; but later he recognised the futility of trying to impose such conditions upon patients and greatly relaxed the rigour of his regime. Finally he treated

each patient individually, varying diet according to the carbohydrate tolerance and calorie requirements much in the manner of Kulz. 30 - 45 calories per kilo he regarded as the optimum for the average adult with an allowance of protein which frequently reached 125 gms. He recognised, however, that patients could get along on 25 - 30 calories per kilo, and when glycosuria was obstinate he did not hesitate to reduce the protein ration to 40 - 50 gms. per day. With Naunyn acidosis became the chief criterion of severity in diabetes. He discovered how necessary it was that carbohydrate be reduced gradually, and he introduced the practice of giving large doses of sodium bicarbonate during this period in "acid" cases. His views on exercise were similar to those of Kulz and he, too, recognised that some patients did best on complete rest. Loss of weight beyond 2 kilos Naunyn considered to be unwise. If the urine was not then free from sugar he advised that the attempt be abandoned. Fast days he re-introduced, finding them helpful in all cases, and frequently very beneficial in cases of acidosis. When coma threatened or when the tinct. ferri perchlor. reaction was marked for three days, fasting was immediately stopped and milk, together with sodium bicarbonate, was administered in large quantities. Meantime fat in the form of butter was withheld as containing many lower fatty acids. This treatment is noteworthy as being that almost universally adopted until over 60 years later when insulin was introduced.

Lenné of Neuenahr seems to have realised more clearly than Naunyn the important part played by protein in the diet;

but he made the mistake of regarding protein restriction as the really important factor, and carbohydrate restriction as of lesser import. To fat consumption he set no limit, considering that appetite and digestion kept this within bounds, and strongly opposing Naunyn's theory that it was the chief factor in acidosis. An occasional starve day he held to be useful but he was by no means enthusiastic concerning this form of treatment.

The latter half of the nineteenth century is notable for the number of "cures" which were introduced. Each of these had its band of adherents, enjoyed a more or less brief vogue, and in turn was replaced by some modification or by an entirely new device. The "Milk Cure" of Karell had a great reputation for a time. In this the diet consisted of 60 - 200 c.cs. of skimmed milk four times daily - an undernutrition dietary with obvious advantages from the viewpoint of to-day. Duncan strongly advocated this type of treatment, and by experiment demonstrated that casein was assimilated more readily than any other protein, and lactose than any other carbohydrate. Strasser employed milk in combination with diet, alternating 3 days of diet with 3 days of milk. Naunyn employed the cure in cases of threatened acidosis, when he considered it useful in warding off coma. Soon sour milk was suggested in place of sweet milk, but van Noorden proved that such milk had to be too bitter before it was definitely helpful, and so curbed enthusiasm in its use. Nevertheless sour milk was very extensively employed, in Scotland at any rate, up to a few years ago.

The "Rice Cure" of von Daring of Amsterdam next enjoyed a considerable popularity. In this case the diet consisted of 80 - 120 gms. of any cereal, generally rice or oatmeal, with 250 gms. of meat, stewed fruit, milk, wine and small amounts of bread. Obviously this was a diet rich in carbohydrate but with a low protein content, and not unnaturally its success was short-lived. It was followed shortly afterwards by the "Potato" diet of Dujardin-Beaumetz. Potatoes were said to possess a peculiar virtue in diabetes by reason of their high potassium and manganese content. Large amounts - 1500 - 3000 gms. - of potatoes alone were given in 24 hours with no other type of food: indeed patients were quite unable to eat anything more, and the improvement recorded must be attributed to this fact. The treatment was extensively practised in France, but it did not obtain much hold elsewhere. In time it, too, followed its predecessors into disuse.

It must be said of Naunyn that he had been profoundly sceptical of the value of any of these "cures". He held strongly that in all of them the essential benefit lay in undernutrition, yet no general recognition was made of the fact that the value of the cure decreased as the quantities of food increased. Instead much work was done on the assimilability of various carbohydrates and particularly in the various types of cereals. As an outcome of this the "Oatmeal Cure" of van Noorden was evolved, a cure which became universally popular

and was widely employed up to the introduction of the "Allen" treatment. The treatment consisted of varying amounts of oatmeal or oatmeal gruel together with eggs and butter. Along with this alkalis, mainly sodium bicarbonate, were given in large doses. Later van Noorden recommended that starve days be introduced. A common practice was to alternate 2 starve days with 2 oatmeal days, alcohol being allowed on the starve days. After a prolonged period of such treatment the diet was gradually increased, only to be dropped again should progress become unsatisfactory, when the process was repeated.

Such were the methods of treatment in vogue at the beginning of the present century. Now would be reported a series of successful cases on one regime, now a series on another; but a close analysis of these records showed that it was the milder cases which responded to almost any form of treatment. Results in the more severe cases were uniformly unsatisfactory. This then was the position in 1914 when the problem was tackled by two sets of workers, one under the direction of Allen in America, and the other under Graham in London. The methods which these observers adopted, and the success which they achieved, fall to be described in another section of this paper.



**METHOD OF DISTINGUISHING TYPE IN CASES OF GLYCOSURIA.**

**((-----))**

Following the discovery by Dobson in 1775 that the sweetness of the urine in diabetes was due to the presence of glucose special attention was directed towards this symptom, particularly with a view to discovering from it an index of the severity of the condition, and so rendering diagnosis and prognosis more accurate. In time, however, it became clear that the significance of this glycosuria varied very greatly. One patient might have sugar in the urine accompanied by thirst, polyuria and loss of weight, and a fatal termination might ensue in the course of a few weeks or months. Another patient might show an even larger amount of sugar in the urine with temporary or entire absence of symptoms of diabetes; and this patient might live for ten, twenty, or thirty years, finally to die of some condition in no way connected with diabetes. Again, a glycosuria occurring in older people was recognised as a much less serious condition than an apparently similar glycosuria in the young; while it was further recognised that there was a glycosuria occurring commonly in patients with arteriosclerosis which seemed to have little or no detrimental effect.

Klemperer and Naunyn both recognised a form of glycosuria occurring at any age, apparently of no significance; but of the condition generally Naunyn<sup>3</sup> rather despairingly wrote "the course of the disease is as variable as can be conceived."

Thus up to quite recent times diagnosis and prognosis in cases of glycosuria were in the last degree uncertain, and time, and time alone, served to differentiate the serious from the trivial.

That to-day we find ourselves more favourably placed in this regard is due to the very rapid advance during the past ten years along the lines of blood sugar investigation, and to the great improvement in the technique of laboratory methods whereby simple and accurate analytical procedures have been evolved. Dobson had noted that the blood serum of diabetics was sweet to the taste, while Ambrosiani and McGregor about 1820 had shewn the presence of fermentable sugar in the blood. To Claud Bernard,<sup>73</sup> however, belongs the credit of performing the first reasonably accurate blood sugar estimations. Generally his results were high, but occasionally he obtained readings around 0.107% which more modern methods have shewn to be substantially correct. Following Bernard numerous isolated observations on blood sugar were recorded, and it was established that in diabetics and in the normal individual after a carbohydrate meal the level was considerably raised. But prior to 1913 no method of blood sugar estimation had been evolved which required less than about 30 c.c. of blood from a vein, and consequently repeated observations on one patient over a short period of time were impracticable. The simplification of the methods

of estimating the sugar in the blood is the outstanding advance in practical medicine which has rendered a differentiation of the various types of glycosuria possible. By these "micro" methods, as they are called, accurate results can be obtained with a few drops of blood procured by pricking the ear or finger, and thus a series of examinations becomes a matter of no moment to the patient.

#### MODERN METHODS OF ESTIMATING THE BLOOD SUGAR.

The first "micro" method of estimating the sugar in the blood to be described was the Bang method, and it deserves special mention on that account; but it has fallen into disuse for many reasons, and further reference need not be made to it here.

The picric acid method of Lewis and Benedict, another early method, required 20 c.c. of blood and therefore need not be discussed. A modification of this method, however, by Cammidge<sup>10</sup> reduced the amount of blood required to 0.2 c.c. and thus brought it into line with the other methods to be described. In this method the blood was collected and laked with water. The protein was removed by precipitation with picric acid and filtering. Thereafter the filtrate was rendered alkaline with sodium carbonate, and boiled for a given time. The solution was then compared in a colorimeter with a standard solution of picramic acid, the

amount of sugar present being read off from a specially graduated tube after dilution with water.

This method I employed from March, 1920 for about two years. It is rendered inaccurate by an excess of creatinine in the blood; and latterly I experienced such trouble in obtaining pure picric acid for purposes of comparison that I abandoned the method altogether. While this method is open to the objections levelled against all colorimetric methods, I consider that I obtained very satisfactory results from its use. Cammidge himself was enthusiastic concerning it, while it was approved of, and extensively used, in the Biochemical Laboratory at Glasgow University.

The Folin and Wu<sup>19</sup> method is deservedly one of the most popular at the present day. Here tungstic acid is used as the protein precipitant, while in the sugar estimation a phospho-molybdic acid reagent is employed. Only 0.2 c.c. of blood are required. The fact that the tungstic acid method for the removal of protein is the most efficient yet devised is a special point in its favour. Mackenzie Wallis and Gallacher, and Calvert<sup>8</sup> have both introduced modifications of this method. In the first (Wallis and Gallacher) the amount of blood collected is estimated by weighing on a torsion balance - a costly instrument: in the second (Calvert) the blood is collected in a platinum capsule and again weighing

is done by means of the torsion balance. I have never employed any of these methods mainly on the ground of expense; but, where this is not a factor to be considered, they are to be recommended.

<sup>38</sup>  
Maclean's method as modified in 1920 is a most satisfactory one. Here again only 0.2 c.c. of blood are required. The blood is added to a solution of sodium sulphate rendered faintly acid, and the mixture is boiled, treated with dialysed iron, and filtered. A measured amount of the filtrate is boiled with an alkaline copper solution containing iodic acid as the inert potassium salt. Later hydrochloric acid is added, when the iodic acid is set free and reconverts the reduced copper into the cupric state. The excess of iodic acid reacts with the hydriodic acid also formed when the hydrochloric acid is added, and free iodine is liberated. The amount of iodine is estimated by titration with a standard thiosulphate solution, using starch as an indicator. The difference in the burette readings between the blood filtrate and a blank control is found, and the blood sugar percentage is calculated from a prepared table.

In this method it is essential that the solutions be very carefully prepared, and that they be fresh, particularly

the thiosulphate. This last is kept in  $\frac{N}{10}$  solution and diluted to  $\frac{N}{400}$  when required for use. It should be renewed every two months. The filtrate and copper solution are boiled for a fixed time, and this difficulty is got over by employing a simple manometer introduced into the rubber tubing to a Bunsen flame.

I have employed this test exclusively during the past two years, and it has proved in every way satisfactory. It loses its accuracy, in my experience, when the blood sugar falls to a low level, as may happen after too large a dose of insulin just prior to, and during, a hypoglycaemic reaction but I have found it accurate to 0.06%, and this is all that is required for general purposes. All the blood sugar readings given in the accompanying charts were obtained with Maclean's method. For the reasons stated above I have thought it wise to discard the Benedict - Camidge results save in the charts illustrating the "Allen" treatment, where they give at least an indication of the progress in these cases..

In the Autumn of 1924 I had an opportunity of seeing the Hagedorn and Jensen method of estimating the blood sugar employed in the Reichshospital in Copenhagen. In this method zinc hydroxide is used to precipitate the protein while

potassium ferricyanide is used to estimate the sugar. Only 0.1 c.c. of blood is required in this method; but, although the results obtained seemed uniformly good, the method appeared to have no advantage over that of Maclean.

#### THE GLUCOSE TEST.

That sugar appeared in the urine of the diabetic in largest amount after a carbohydrate meal had long been known. It followed naturally that estimations of the sugar in the blood were made by the earlier observers before and after such a meal in all cases of glycosuria, and the invariable rise in the blood sugar level was demonstrated. Where a series of observations were made it was noted that the blood sugar reached a higher level in some cases, and remained high for a longer period, than in others, and it thus became clear that by such a series of estimations following the ingestion of a constant amount of carbohydrate a distinction could be made between the various types. The test which was gradually evolved is known as the "glucose test". It is carried out as follows:-

Three and a half hours after the last meal - or preferably before any food has been taken in the morning - the patient passes urine and a blood sugar estimation is made. Then 50 grammes of glucose in 100 c.c. of water are given as a draught. Thereafter blood sugar estimations are made at



half hourly intervals for two hours, and samples of urine are obtained at one hour and two hour intervals.

Occasionally it may be necessary to continue the blood sugar observations to two and a half to three hours, but this is rarely essential. The curve obtained by graphing these results is called the "Glucose Curve".

### GLUCOSE CURVES IN THE NORMAL SUBJECT.

It is necessary that the effect of such a test on the normal individual should first be considered briefly. The initial (resting or starving) blood sugar level lies between 0.08% and 0.11%, generally about 0.10%. Half an hour after glucose it has risen to 0.15% to 0.18%: in no case does it ever transcend the latter figure. Gray<sup>23</sup> in a series of three hundred cases found it very rare to get a reading over 0.16%. Folin and Berghund<sup>18</sup> working with a number of medical students obtained distinctly lower readings than Gray, and this they attribute to the absence of emotional disturbance. Cammidge records a case in which at a first glucose test on a very nervous subject the blood sugar at half an hour reached 0.21%: but subsequent tests on the same individual shewed him to be entirely normal. This nervous factor has to be considered, not only during the glucose test, but more frequently in the level of the initial blood sugar, a point to which further reference will be

made later. The important point, however, is that never in the normal case - the neurotic case quoted above cannot be considered as completely normal - does the half hour blood sugar rise above 0.17% or 0.18%.<sup>33,9.</sup> Maclean regards the second figure as more correct, but Gray, Folin and Berghund, and Joslin<sup>31</sup> favour the first, viz:- 0.17%, and, in the charts which follow, that reading is marked as the upper level of normal blood sugar excursion.

At one hour the blood sugar reading has fallen, and is generally in the region of 0.13%; while one and a half hours after glucose in the great majority of cases it has returned to the normal fasting level. Invariably in the normal healthy individual under sixty the blood sugar level is normal in two hours. Thus the characteristic normal glucose test curve shews a rapid rise to a level never above 0.18% followed by an almost equally rapid fall, and a return to the resting blood sugar level within two hours.

Chart I. curve (a) shews a typical normal "glucose curve" and, while such a curve is that most generally obtained, there are minor variations in its form which are quite compatible with the normal. Thus in curve (b) the top of the curve is flat. It is almost certain that in this case the highest blood sugar level has been reached at about three quarters of an hour after glucose, and that the half hourly and one hourly readings are points upon an ascending and descending phase respectively. Curve (c) is that obtained from an apparently healthy man of sixtyfive. Here the return to normal is less rapid than in curve (a), but the normal level is reached in two and a half hours. Such a delay in the return to a normal level is not uncommon in elderly people in whom, indeed, I have come to regard it as the normal type of curve.

GLUCOSE CURVES IN RENAL GLYCOSURIA.

In an occasional case of glycosuria one may find that the blood sugar curve following 50 grammes of glucose conforms entirely to the normal in form, but that, while sugar is absent from the urine before the glucose is given, it appears in the specimen taken at one hour, and perhaps again in that at two hours. It is rare, however, to find more than 2% of sugar in the one hour specimen, or more than a trace after two hours. In my experience this type of case is uncommon, but it does occur, and is of particular interest when treatment with insulin is considered, as will be explained later. This condition is known as "Renal Glycosuria". Chart II illustrates three glucose curves of this type together with the urinary sugar estimations. ~~In no case does the sugar in the urine exceed 2%.~~

LAG GLUCOSE CURVES.

Another and a more common form of curve is that to which the term "lag curve" has been given. Such a curve is illustrated by chart III.

The resting blood sugar reading here is normal. Half an hour after glucose the reading is above 0.17%, in this case 0.2%. At one hour the curve has fallen to a considerably lower level (0.16%) while at one and a half hours the normal level is reached. In this curve then we have a rapid rise from the normal to a level above 0.17% followed by a rapid fall to normal, quite comparable with that found in the two types of curve just described. The urine is sugar-free before glucose, contains 2.5% of sugar at one hour, and a faint trace at two hours.

Chart IV. illustrates a series of "lag" curves which I wish briefly to consider.

Here the initial blood sugar is normal in the majority of cases. In a few, however, it is a little above the resting level, as in curves 4, 7, 9 & 10. As I have said above, this is often due to emotional disturbance, which may either of itself raise the blood sugar level, or may considerably interfere with the absorption and storage of the carbohydrate of the previous meal. Again, in private practice, while all are warned against eating anything for four hours before the test is to commence and to take then only a light meal, it is difficult to rule out the possibility of some not readily assimilated food having been eaten. Provided, however, that the initial blood sugar reading be not over 0.14% it may be regarded as normal. Half an hour after glucose the reading obtained is above 0.17% - in the cases here illustrated the lowest half hour reading is 0.182% and the highest is 0.245%. Curve 9 is an exception to which further reference will be made later. One hour after glucose the curve has fallen to a considerably lower level. In curves 1 - 5 the one hour reading is below 0.17%; in 6 - 11 it still lies without the normal limit. Estimations at one and a half hours after glucose are normal in 1 - 5, but in 6 - 11 they are still above the normal resting level, although within normal limits. At two hours, however - and this is the important point - the blood sugar readings in all

ten cases are normal.

The corresponding urinary sugar results now demand consideration. In all cases, with the exception of No. 7, sugar was absent from the urine before glucose was administered. One hour later sugar was invariably present, sometimes merely a trace as in 1, but sometimes to as much as  $\frac{5}{100}\%$  as in <sup>11</sup>2. In five cases the urine was again sugar free at two hours; in three the glycosuria persisted but in smaller amount. In nine the patient was unable to pass urine after one hour and only a two hour sample was available.

Here we have a series of curves whose first and final readings are similar to those obtained in normal individuals, but one at least of whose intervening readings is without the normal limit. Associated with these hypernormal readings there is a glycosuria of varying degree.

THE DIABETIC GLUCOSE CURVE.

The last type of curve is the Diabetic Curve. In this group we have to distinguish between the results obtained from a glucose test in (1) a diabetic under treatment, and in (2) an untreated case.

Chart V. curve 1. shows a typical result in a case undergoing treatment. The resting blood sugar reading is normal but the subsequent readings are all above the normal limit, and - again the point to be emphasised - the last reading, two hours after glucose, is far above the initial one. With regard to urinary sugar, there is merely a trace in the specimen taken before the test, but the one and two hourly specimens contain sugar in amount.

Of the results obtained in an untreated diabetic curve 2 is an example. The initial blood sugar is high, but the level is raised even higher by the glucose, while the fall at the expiry of two hours is negligible. Glycosuria, marked before the glucose is given, is further increased by the test.

In these cases, with the exception of the first blood sugar estimation in curve 1. all readings are above the normal. There is no real attempt at a return to normal levels, and this point demands a brief elaboration. As has been pointed out above, the normal blood sugar level never rises above 0.18%. No matter how much glucose be given - and 200 grammes have frequently been administered



in a single dose - the blood sugar cannot be forced above this level. Should in any case the blood sugar rise above this level then sugar will appear in the urine, and consequently it has been called the "normal renal threshold".

Now it has been shewn that in the normal case a rapid fall occurs in the blood sugar level, so that, if no further carbohydrate be given, the resting level is very quickly reached. This must mean that somewhere below 0.17% some mechanism comes into play whereby the sugar is rapidly withdrawn from the blood, and that, in the abnormal cases, this mechanism is in some way disturbed. Two suggestions seem feasible. Either there is a sudden and rapid breaking down of sugar in the circulating blood, or at a certain degree of saturation of the blood with sugar a storage mechanism is brought into play, possibly a storage of sugar in the liver in the form of glycogen.<sup>67</sup> The bulk of evidence goes to support this second theory, but a discussion of the matter lies without the scope of this paper.

Assuming the correctness of this suggestion let us consider the various types of curve described above. The "Renal" type of curve differs in no respect from the normal, but sugar is present in the urine. This glycosuria occurring after ingested carbohydrate is now almost universally regarded as being due to some abnormality in the kidney itself whereby sugar is permitted to escape into the urine, although

its concentration in the blood is not abnormally high. In other words the "renal threshold" is lower than normal. There is no defect in the sugar disposal mechanism.

In the "lag" type of curve the highest reading, almost invariably that obtained half an hour after glucose, is above 0.17%. During the period in which the blood sugar level remains above this figure sugar escapes into the urine in greater or less amount. The fall in blood sugar, however, is rapid and quite comparable with the normal. This would suggest that the mechanism for disposing of the circulating sugar was not stimulated into activity until the blood sugar had reached a higher level than in the normal, but that, once having commenced, it proceeds with normal rapidity. The idea of delay in initiating the storage process is conveyed in the term "lag".

The diabetic curve requires little comment. It begins either below the renal threshold when sugar is absent from the urine, or above the threshold when sugar is present. The later readings are all well above the threshold level and glycosuria is pronounced. Here the storage mechanism is difficult to stimulate to activity, and, even when begun, it is very inefficient. This defect in the sugar disposal mechanism, as shewn by the failure of the curve to return to normal within two hours, is the characteristic feature of the diabetic curve.

THE SIGNIFICANCE OF VARIOUS TYPES OF  
GLYCOSURIA.

"Renal Glycosuria" is almost certainly of no significance. Indeed, as will be apparent in case III. strict dieting in such cases leads only to loss of weight and general ill health. So far as my experience goes these patients can be allowed an unrestricted diet.

The diabetic cases undoubtedly require treatment in more or less rigorous form. The glucose test is sometimes very useful in such cases for purposes of prognosis, a group of lower readings being generally a more favourable sign than a group at a higher level. In the great majority of diabetics, however, it is unnecessary to perform a glucose test. Thirst, polyuria, loss of weight, and glycosuria establish a diagnosis without further confirmation being required. In this connection Joslin<sup>31</sup> has drawn attention to a statement made by Allen concerning the administration of glucose to depancreatized dogs. Allen states - "In the early stage glucose is more powerful than starch in producing diabetes, and animals which are progressing toward complete recovery on starch diet can be sent into hopeless diabetes by admixture of glucose". In view of this pronouncement from so eminent an authority it would seem wise to refrain from performing the glucose test upon cases of undoubted diabetes. It should be reserved for diagnostic purposes in doubtful cases.

The cases which present most difficulty are those which exhibit a "lag" response to the glucose test. Maclean<sup>39</sup> regards these cases as being on all fours with the "renal" type of case, and he permits ordinary diet in all patients who present this type of curve. Personally I have always treated the "lag" case with greater caution. A reference to chart IV will help to make clear my reason for adopting this attitude. In curves 1 to 5 only one reading, that at half an hour after glucose obtains above the normal renal threshold. In curves 6 to 10 two readings are above this level, while in curve II all save the first and last readings are above 0.17%. Indeed it is only the clear evidence of a return to normal at the end of two hours which establishes these cases as being of the "lag" type. If now we compare this last curve with one of those on the diabetic chart - say No. 7 - we are struck by the fact that only in the level of the two hour blood sugar reading does the distinction lie. One is naturally tempted to ask:- Is it justifiable to condemn one patient to a life of restricted diet, and to the inconvenience and discomfort which this must entail, and to assure another that he may eat what he likes on the evidence of this single blood sugar estimation? Consideration must be given to the following cases before attempting to answer this question.

Chart VI. I have labelled ? Diabetic.

It represents five curves whose interpretation is difficult. Curve (1) is a "lag" curve save that the two hour reading has risen to 0.17%. This phenomenon I have seen occasionally in hospital cases, and I can find no explanation for it. Professor Adam Patrick told me that he, too, had encountered the same curious ending to the curve in two "renal" cases, and that he had been unable to account for such a rise. So far as my limited experience goes it would seem to be of no importance; but taken in conjunction with the comparatively copious glycosuria in this particular case it causes it to remain under suspicion.

Curve (2) would be regarded as normal did the final blood sugar fall to the normal resting level. The absence of glycosuria supports this view, but here again, with an apparent delay in the storage of sugar, one is not satisfied that the subject is entirely normal from the result of this test.

Curve (3) shows the result of a glucose test performed on the same patient as curve (2) but two weeks later. This curve is of the "lag" type save that the initial and final readings are high. A trace of sugar appears in the urine at one hour. Again the curve is inconclusive, but the failure of the two hour blood sugar to return to normal in both glucose tests is very suggestive of a potential diabetes.

Curve (4) is another doubtful "lag", very similar in

every respect to (3); but although the two hour estimation is lower the other readings are at a higher level, while the glycosuria is more marked. This case, too, is more than likely to be an early diabetes.

Curve (5) is that obtained in a man of 55 years. I regarded this as a "lag" curve, the return to normal being delayed as is so common in more elderly people. Unfortunately it was not possible to repeat the blood sugar test at two and a half hours, but I am confident that it would then have been 0.10% or 0.11%. That this was almost certainly the correct view is proved by this man's condition to-day, a year later. He is in excellent health, free from glycosuria, the result of a very moderate restriction of carbohydrates.

These examples serve to show how difficult it may be to interpret aright the results of the glucose test, particularly in regard to "lag" results. The following two cases illustrate this point even more clearly :-

M.L., a well nourished girl, aged fifteen, was admitted to the Royal Infirmary on 5/2/24 complaining of thirst and polyuria which had come on suddenly three weeks before. She had been employed for six months in a baker's shop and had eaten sweetened cakes in large numbers during that time. The family history was negative. The urine contained 3% sugar. Glucose tests were performed as shewn in chart VII. The first test was made after a brief period of starvation, but from that time onward the diet remained constant at C.80 P.60 F.120. The

first and second glucose tests gave typical diabetic curves with urinary sugar results to correspond. The fourth and last tests gave curves which, with the urinary sugar observations, would suggest a "renal" glycosuria. All tests were made in the early morning before any food had been taken. Six months after dismissal from hospital she returned for a further test (No. 6). The curve was now typically diabetic.

J.O., a boy aged 6, was admitted to hospital on 7/9/22 complaining of headache, thirst, and frequency of micturition of some two and a half months' duration. While in hospital there was never more than a trace of sugar in the urine, and his blood sugar, with one exception, never exceeded 0.15%. Chart VIII shows the result of a glucose test at this time. The curve might be called a "lag" though no sugar was detected in the urine. Certainly the return of the blood sugar to normal in one and a half hours is satisfactory. Less than a year later the boy was re-admitted to hospital, and now insulin was necessary to keep the urine sugar free. Curves 2 and 3 show the results of glucose tests at this time, and are clearly diabetic. The boy had not been kept on strict diet during the intervening period.

From a consideration of these results it will, I think, be clear that it is unwise to make a diagnosis in many cases, and dangerous to do so in doubtful "lag" cases, on the result of a single glucose test. Two or more observations should be made at intervals of some weeks before a definite opinion is

pronounced, and especially before permitting an unrestricted diet. In younger patients where it is essential that a correct diagnosis be arrived at as soon as possible I generally repeat the test at fortnightly intervals, keeping the patient upon a fixed diet throughout; but in older patients, fit and at work, I have found it very satisfactory to repeat the test at, say, three monthly intervals. I shall quote in some detail three cases to illustrate the results to be obtained by this practice.

CASE I : R.T. aged 18, a strongly built lad who had just left school, was found to have sugar in the urine on routine examination for insurance. He was sent to me to determine the significance of the glycosuria. He felt perfectly fit and played football regularly every saturday, in addition to playing Badminton vigorously twice a week. His physique was excellent. I could find nothing abnormal on physical examination. He had never been ill save for measles and chickenpox in childhood. The family history was good.

Chart IX. illustrates the results of three glucose tests performed on this patient. The first glucose test has been referred to above. It was a doubtful "lag" curve, the return to normal being unsatisfactory. Sugar was present even before glucose was given, which made the circumstances rather more suspicious. Nineteen days later the test was repeated, the patient being allowed everything save obvious sugar, (sugar, jam, icing, &c.) in the interim. The second test gave a rather



unexpected result. The curve here would be regarded as entirely normal had the final blood sugar reading reached the normal resting level, while the absence of sugar in the urine throughout the test would have confirmed this opinion; but with a similar apparent defect in storage as in the first test it did not seem prudent to regard the glycosuria as being negligible.

Two weeks later a third test was made, the diet having remained unchanged since the first test. The third curve must be regarded as of a mild diabetic type, a view supported by the appearance of sugar in the urine at one hour. This case was therefore diagnosed as a potential diabetic, and a restriction in the carbohydrate of the diet was advised.

CASE II : A.B. aged 36 years, a busy professional man, first consulted me concerning a glycosuria which he had known to be present for eleven years. In 1916 he had contracted dysentery in Palestine, and this had become chronic. A year later a bacteriological examination of the fæces was made, and the bacteriologist suggested that, with the organisms found, the patient was likely to have a glycosuria. The urine was tested and sugar was discovered. Following the discovery of glycosuria he was treated from time to time with vaccines, but the condition persisted. He remained well throughout the entire period save for occasional attacks of diarrhoea. He desired glucose tests merely to see if further light could be thrown upon the significance of his glycosuria.

Patient had never had any illness save childish ailments prior to his dysentery. The family history was good.

He was a strong, sparely built man of healthy appearance. Nothing abnormal could be found on physical examination. He had dieted in a moderate way for many years. Glucose tests were performed on 13/6/23, 5/12/23 and 3/7/24.

From the accompanying chart, (chart X) it is clear that, save for the rather high initial reading, the first curve was a typical "lag" with urinary sugar readings to correspond. I advised an increased diet, avoiding only obvious sugar (sugar, jam, &c.) and upon this diet he remained for six months. A second glucose test at this time No. II) gave a frank "lag" curve again. The patient was now permitted to eat what he

wished, and six months later the third test was carried out (No. III). This test showed a very slight "lag", a considerable improvement on the two previous results. He was in excellent condition and felt much more able for his work than formerly.

I met this man quite recently and was struck by the improvement in his appearance. He told me that he had refrained from visiting me again lest a further test should lead to alteration of a regime on which he felt more fit than he had done for years. Not only did he feel better physically, but he was much happier and more contented; and this he attributed mainly - and I think quite rightly - to the removal of dietetic restrictions.

In this case the repeated tests have shewn that the type is undoubtedly a "lag" and that increased feeding has been of benefit to the patient, even as shewn by the glucose test curves. The glycosuria would appear, therefore, to be of no significance. Here the test has been extremely helpful from every point of view.

CASE III : R.M. aged 18, a clerk, first consulted me on 9/3/23 concerning a glycosuria discovered at examination for insurance. He had been sent by his own doctor. For himself he made no complaint.

He had measles and scarlet fever in childhood. His father was known to have had glycosuria for twentyfive years, but it never inconvenienced him so far as he knew. Otherwise the family history was negative.

He was a youth of spare build and fresh complexion. His weight was 8 st. 12 lbs. All the organs were healthy so far as I could discover. The urine then contained 4.3% sugar, but no acetone.

The first glucose test was performed on 14/3/23 when a fairly typical lag curve was obtained (chart XI) The rather high pre-glucose blood sugar reading was attributed, as explained above, to nervousness. The urinary sugar curve, however, was very disconcerting, and with so high a reading as 8% at one hour I was afraid to act upon the blood sugar results. The patient was therefore placed upon a much restricted carbohydrate diet - 60 gms. of bread, 30 gms. of potatoes, and green vegetables ad.lib. Six weeks later he weighed 8 st. 8 lbs. He complained of general weakness and of feeling unfit for his work. Certainly he did not look so well as when first examined. A glucose test now gave a normal curve with that curious rise in the final estimation to which reference has been made above. The urinary sugar

was distinctly less than on the first occasion, the largest amount being 3% at one hour. The carbohydrate of the diet was now increased to 120 gms. of bread, 60 gms. of potatoes, with fruit and vegetables ad.lib. Two months later the weight had remained unchanged and, while he complained less of general weakness, he was unfit for the moderate exercise (golf) which he was accustomed to take. The diet was now altered to full diet without sugar or jam, but with no other restriction. Three months later he was looking and feeling very fit, and the weight had risen to 8 st. 11 lbs. Ten months later (8/7/24) a third glucose test was performed. Again a normal blood sugar curve was obtained, and again the amount of sugar in the urine was considerable. The patient, however, now weighed 9 st., appeared in excellent health, and stated that he felt perfectly well.

This case is peculiar in several respects. In the first place he appears to have changed from a "lag" into a "renal". Possibly nervousness accounts for the high blood sugar reading in the first curve; almost certainly the second and third curves represent his true condition - a renal glycosuria. The amount of sugar passed in the urine, too, is unusually large. This must mean that the "renal" threshold in this case is very low: indeed, from the fact that sugar is present in the urine even before glucose, one is justified in assuming that the "renal" threshold is below the resting blood sugar level,

i.e. below 0.10%, the lowest threshold which I have met with in any case of glycosuria.

Another interesting point is the presence of a glycosuria in the father, evidently of no significance. It has been present for twentyfive years and he has never restricted his diet in any way, yet he has remained in excellent health during this time. The son would seem to belong to that group of familial renal glycosurias at present the subject of investigation.

This case illustrates very clearly the harm which may be done by restricting diet in these cases. The glucose test and general condition following a year of practically unrestricted diet show that this line of treatment is sound, although further observation will be necessary before one can be perfectly certain in this regard.

This group of cases illustrates how the difficulty in classifying these atypical cases may be, to some extent, overcome; but such cases should never be lost sight of even after a definite line of treatment has been decided upon. Sufficient time has not yet elapsed to allow of authoritative information being made available concerning treatment based on glucose tests, and, personally, I shall continue to watch with interest the cases which I have here quoted.

"RENAL" CASES.

Mrs. X. aged 28, a healthy-looking, strongly built woman, was sent to me to investigate the significance of a glycosuria which had been present for three years. The glycosuria was discovered on a routine examination of the urine when the patient was confined to bed with a slight chill. She had been married for four years but there was no family, her doctor having advised against pregnancy lest the glycosuria be aggravated. She appeared in perfect health and her own doctor stated that she was physically sound. I did not examine her.

Chart II No. 1 shows the result of a glucose test on this patient. The blood sugar curve is normal in every way while only a trace of sugar is present in the urine at one hour - a typical "renal" glycosuria. This result was not unexpected as this lady had never dieted and had never made any complaint of illness.

Since the test was made this patient has had a perfectly normal pregnancy and a healthy child. Her health remained excellent throughout, and to-day her doctor reports she is in excellent condition.

This case illustrates how a glucose test may allay the fears of an over-anxious practitioner, and, at the same time, lead to a much more satisfactory life for the patient.

Nos. 2 and 3 have already been referred to. (Case III above)

"LAG" CASES.

X.Y. aged 56, a Chemist, consulted me concerning a glycosuria which he had discovered accidentally while using his own urine to test the condition of some Fehling's Solution which had been lying in his shop. The test had been positive with the old Fehling, but he discovered subsequently that the same result was obtained with new solution. He was greatly concerned about his condition and asked me to investigate it. He made no complaint of feeling unwell.

He was a well-built man, looking younger than his years apparently in excellent health. Save that his arteries were slightly thickened I could find nothing abnormal in his physical condition.

Chart IV. No.(3) shows the result of a glucose test - a typical "lag" curve. The urine was sugar free before glucose and at two hours, but there was 0.5% of sugar at one hour.

With the blood sugar curve showing so complete a return to normal (both one and a half hour, and two hour estimations were satisfactory) one felt confident in re-assuring this man and giving a good prognosis. He was very much relieved. He has voluntarily restricted his carbohydrate even more than was suggested to him, and since the date of the test, fifteen months ago, he has been very well. Indeed to-day he states that he feels more fit for work than he has felt for many years past.



W.B. aged 51, a Derbyshire Farmer, was sent to me for investigation as to the significance of a glycosuria which had been discovered on routine insurance examination, and concerning which he had consulted a homœopathist. He appeared healthy, and it was reported to me that nothing had been discovered on physical examination, and that the family history was negative. I did not examine him.

Chart IV. No. 5 curve gives the result of the glucose test. The first, fourth and last blood sugar estimations were normal; the half hour reading was 0.243%, the highest obtained in this series. The urine was sugar free before glucose, contained 0.5% sugar at one hour, and was again sugar free at two hours. He was reported upon as a typical "lag". I have no information concerning his treatment or subsequent progress.

N.Mc. aged 34, was seen by me first on August 16th, 1923. He had been operated upon four days previously for an appendix abscess, and the presence of sugar and acetone in the urine caused the surgeon to ask me to see him with a view to giving insulin if considered necessary. The insulin treatment of this case I shall deal with in a later section. It will suffice to say here that he made a slow recovery, and two months later the appendix wound was still discharging slightly. He had been on holiday, and a local doctor to whom he had submitted occasional samples of urine had reported the constant

presence of a trace of sugar. As he wished to be insured as soon as the wound was healed he desired to have a glucose test performed so that he could meet any objections which might be raised should sugar be present at his examination. He was sure himself that the glycosuria was negligible, and he desired confirmation.

He was a man of excellent physique. I have seldom seen a man of finer build. Even after his long illness he appeared to be in robust health. Save for the imperfectly healed appendix scar physical examination was entirely negative. The family history was also negative.

The glucose test result is shewn in chart IV. No. 6 - a typical "lag" curve. The urine was sugar free before glucose. At one hour it contained .055%, and at two hours a trace of sugar. Shortly afterwards he was examined for insurance and sugar was found in the urine. A report was submitted on request, but in spite of a favourable prognosis he was refused.

To-day sugar persists in small amount as before in the urine. He refuses to restrict his diet in any way, but he looks and feels in excellent health.

W.S. aged 58, a Commercial Traveller, was sent to me for investigation. He had complained to his doctor of weakness in the legs, and routine examination had revealed a glycosuria.

He was a sparely built man who looked older than his years. There was evidence of some bronchitis with slight emphysema. The arteries were thickened. The knee jerks were present. His sight was excellent with suitable glasses.

Chart IV. No. 8 shows the result of a glucose test performed on this patient. The curve is a typical "lag", showing the more slow return to normal which is common in older people. The urine, free from sugar before glucose, contains 1.3% at one hour and 0.5% at two hours.

A diet without sugar and jam, together with a slight restriction of other carbohydrates, was advised as investigation proved that he was much addicted to sweet foods. As the urine was very acid an alkaline mixture was also prescribed. To-day - fifteen months later - his doctor reports that sugar quickly disappeared from the urine, but that it has returned in small amounts from time to time as a result of lapses in diet. He has been more fit for work during this period than before, and the feeling of weakness has gone from his legs. To-day he is in excellent health.

A.B. aged 42, a Dumfriesshire Farmer, was sent to me for investigation, sugar having been discovered in his urine on routine examination for insurance. It was reported to me that no other physical defects had been discovered.

He was a powerfully built man apparently in excellent health. He stated that he had never been ill so far as he could remember. The family history was negative.

The result of the glucose test is given in chart IV. No. 9 and shows a typical "lag" curve. The initial blood sugar reading 0.122% I attribute to nervousness.

J. McL. aged 58, a Traveller, was sent to me for investigation of a glycosuria which he had known to be present for one year. He had consulted his doctor at that time on account of a progressive loss of weight, and frequency of micturition. Following a period of fairly strict dieting these symptoms had disappeared, and, save for an occasional boil around the buttocks, he had been very well ever since. The diet latterly had been much less restricted, but even that had become irksome to him, and he now desired to know if he could return to ordinary food.

He was a stoutly built, healthy looking man who appeared younger than his stated age. I did not examine him, but his doctor informed me that he had found nothing incompatible with his time of life on physical examination.

Chart IV. No. 10 illustrates the result of the glucose test - a typical "lag" curve with, again, a slightly raised pre-glucose blood sugar reading. The urine was sugar free before glucose, contained 0.5% at one hour, and at two hours contained merely a trace. The small amounts of sugar are in striking contrast to the high half hour and one hour blood sugar readings. Almost certainly in this case the "renal"

threshold is above the normal 0.17%, probably around 0.2%.

While from these findings there seemed no adequate reason for a severe restriction of the diet, a consultation with his doctor revealed the fact that he was a perfect glutton for sweets of all kinds, and consequently it was considered advisable to carry him on as before. The fact, too, of his having had such significant symptoms as polyuria, furunculosis, and loss of weight made this seem all the more necessary. Since the test was performed six months ago the patient has been very fit. His doctor states that he frequently breaks diet, but, as this probably brings him at these times on to a diet which originally he might safely have taken, the policy adopted seems to have been a wise one.

J.H. aged 28, a Motor Driver, complained of loss of appetite and increasing dimness of vision. He had not felt really fit for the past two years.

He was very thin and poorly nourished. There was evidence of a lesion at the right apex, probably tubercular, but this was apparently quiescent. No other abnormality could be detected on physical examination.

The result of a glucose test is shown on chart IV. No. 11. The curve must be classed as a "lag", but it is atypical in that the half, one, and one and a half hour readings are all above the "renal" threshold. (This curve has been discussed above in relation to the diabetic curve). A moderately restricted carbohydrate diet was advised. Three months later the test was repeated when a typical "lag" curve was obtained. The man was looking much better, and had put on a little weight.

DIABETIC CASES.

K.K.P. aged 43, a Minister, consulted me regarding a glycosuria which was discovered on examination for insurance. He made no complaint, had never been ill, and regarded himself as particularly fit when he presented himself for this examination. I had known him for many years and to me he appeared as well as I had ever seen him. He was rather thin, but of good physique, I did not examine him, but the insurance doctor reported that he could find nothing save the glycosuria on physical examination. The family history was negative.

Chart VI. No. 4 shows the result of the glucose test. The initial reading of the curve is high, more high than one would expect as the result of nervousness; and moreover I regarded the nervous factor as negligible in this patient, a very self-possessed man. The return to resting level is also unsatisfactory, the reading at two hours being as high as 0.14%. Meantime the urine was sugar free before glucose and at two hours, but showed a trace of sugar at one hour. I regarded this test as inconclusive as I have pointed out on page 22; but I reported on the results obtained that I regarded him as a potential diabetic and suggested that the test be repeated. However, he was accepted for insurance, and consequently no second test was performed. I have not seen or heard of this patient since that time.



C. O'H. aged 58, a Merchant, consulted his doctor on account of persistent headache. Sugar was discovered in his urine on routine examination, and I was asked to investigate and treat his condition.

He was a small man, somewhat obese, but active. There were no skin lesions. The knee jerks were normal. The pupils were equal, and reacted to light and on accommodation. Heart and lungs were normal. The arteries were healthy. He had worked very hard for many years, had long hours, never took a holiday, and took his meals at very irregular times. He was very constipated. The family history was negative.

With a few days rest in bed and with correction of the constipation the headache disappeared completely. At the same time a slight restriction was placed upon the carbohydrate of the diet - he was allowed three slices of bread and one potato per day - and the sugar rapidly disappeared from the urine. As he was very anxious concerning the glycosuria I performed a glucose test.

Chart VI. No. 5 shows the result of this test. The curve would be regarded as a "lag" but for the fact that the return to resting level is slightly delayed, and the half hour reading is abnormally high. In addition to this the urine, while sugar free before glucose, shows 2% of sugar at one hour and 2.5% at two hours.

With these findings, and as he had now become accustomed

to the idea that sugar was bad for him, it was decided to adhere to the mildly restricted diet mentioned above. He was advised also to cut down his work, to take his meals regularly, and to avoid constipation. I had the opportunity of seeing this patient again quite recently with a mild sciatica. Fourteen months of this diet has left his weight unchanged, but he feels more fit than he has done for some years. There has been no return of the headaches, and the urine is sugar-free.

I am inclined to think that it would have been more correct to regard this curve as a "lag" with the imperfect return to resting level in two hours commonly found in elderly individuals. If this view is correct it makes no material difference to the treatment, but the prognosis is distinctly better.

H.A. aged 52, a Clerk, was sent to me for investigation. Sugar had been discovered in his urine two years previously during a sharp influenza, and following this discovery he had been carefully dieted by his doctor. As a result he had felt so well that he had expressed his intention of returning to ordinary diet, but, on his doctor's advice, he had seen a consultant before taking this step. The consultant sent him to me for a glucose test.

He was a thin, pale, rather nervous man. I did not examine him, but the consultant informed me that he could find nothing definitely wrong on physical examination.

Chart VI. No. 7 shows the glucose curve in this case. The patient was still on the diet which had kept his urine sugar free when this test was performed, and he had had no food for five hours. The initial reading was normal, but the remainder were all above the normal level and the return, as is apparent, was very imperfect. The urine was sugar free before glucose, but showed 0.5% of sugar at one hour, and 2.5% at two hours.

This result proved that this patient was a very mild diabetic, and indicated that a return to ordinary diet would be extremely unwise. The patient was quite satisfied that dietetic restrictions were necessary, and he has continued on the original regime since the date of the test one year ago.

T.W. aged 59, a Country Gentleman, had first complained to his doctor of dryness in the mouth a month before I saw him. The doctor had found sugar in the urine and had placed him upon a restricted diet. This had speedily proved irksome to the patient, and he insisted upon a more exhaustive investigation to make sure that such treatment was necessary.

He was a tall, strongly built man of healthy appearance. The skin was clear. The knee jerks were present, but elicited with difficulty. The heart was much enlarged: the sounds were free from murmur: the second aortic sound was accentuated. The arteries were sclerosed and tortuous: the blood pressure was 225 mm. Hg. The lungs were clear. The abdomen was protuberant, but nothing abnormal could be found on examination. The urine contained albumen in amount. He had consumed a large quantity of alcohol for many years. Albuminuria had been known to be present for three years. The family history was negative.

Chart VI. No. 8 shows the result of the glucose test in this case. The initial blood sugar reading was high, and after glucose the level rose to a maximum of 0.325%. At two hours the original level had not been reached. Meantime sugar appeared in the urine in amount in all three

specimens.

This patient was obviously a diabetic and clearly treatment was not being sufficiently rigorously enforced. The patient realised as the result of the test that more stringent measures were necessary and promised to carry these out. I heard subsequently that he fulfilled this promise, and that he was very well and free from glycosuria on a moderately restricted diet.

T.T. aged 53, a Company Director, was sent to me for investigation and suggestions as to treatment. Ten years previously he had suffered from boils, and when his urine was tested at that time it was found to contain sugar. His doctor placed him upon a diet of limited carbohydrate, and to this diet he had remained faithful, with occasional lapses during all these years. When the diet was rigidly enforced the urine was sugar free, but he never felt any ill effects when he broke diet and glycosuria returned. It was only the continual promptings of his wife and his doctor which kept him on the proper lines. Curiously enough he was morbidly afraid lest any of his friends should suspect him of having diabetes. I think he believed it to reflect adversely upon his moral character, although he would never be definite on that point. However, he had adopted all along the most elaborate precautions and subterfuges to prevent the fact that he dieted being known, and he had returned to his doctor because this manner of life was becoming daily more irksome. He was most anxious to know if he could return to normal diet.

He had never been ill so far as he could remember, nor had he ever complained of thirst, polyuria, weakness, or loss of weight .

He was tall, stout, and florid of complexion. There were no skin lesions. The heart and lungs were normal. The arteries were remarkably soft, and the blood pressure was 145 mm. Hg. The knee jerks were absent. A preliminary

examination of the urine showed Sp. Gr. 1024, sugar 2%: acetone nil, albumen nil. The blood sugar was .306% fasting.

The patient was assured that a glucose test was unnecessary, but he insisted. The result is shown in chart IV. No. 9. The initial and lowest reading is 0.28%, rising in half an hour to 0.36%, and thereafter more slowly to 0.40% at one and a half hours. The fall in the curve is negligible. Sugar was present in all three specimens of urine. In the third it amounted to 6.2%.

The patient was very much disappointed with this result, but he was also quite satisfied that dieting was indeed necessary, so that from this point of view the test was decidedly useful. The continuance of his former diet was advised with a few slight modifications to give variety. His doctor reports that he has remained sugar free for the past year, and that to-day he is in excellent health and fit for his work.

It will be noted that the resting blood sugar in this case was very high (0.28%) even for an elderly patient, particularly when under treatment. In face of this it would appear wrong that he should be advised to continue on the same lines; but there are many factors to be taken into consideration with regard to this point, and with these I shall deal in a later section. I wish merely to emphasise here the value which the glucose test may have in satisfying a patient as to the necessity for treatment.

L.B. aged 34, an Engineer, was sent to me in May, 1923, for report concerning his suitability for treatment with insulin. In September, 1922, he had gone to his doctor complaining of excessive thirst, and examination of the urine at that time had revealed the presence of sugar. He was dieted in a very general way with the result that his discomfort rapidly disappeared, and he was able to continue at work throughout the winter. In the spring, however, thirst returned accompanied by polyuria and weakness, and I saw him as he did not now seem to respond to dietetic treatment.

He was a thin, muscular man, 8 st. 12 lbs. in weight. Physical examination was entirely negative. The urine contained 7.5% of sugar.

A brother-in-law suffered from diabetes, but there was no history of glycosuria in his own family.

A glucose test was performed on this patient and the result is shown on chart VI. No. 10. The pre-glucose blood sugar reading is 0.44%, a surprisingly high figure three and a half hours after food in a comparatively young man fit enough to be at work. The effect of the 50 gms. of glucose was even more remarkable. The blood sugar at half an hour was 0.43% and thereafter all readings were lower, the last being 0.38%. The corresponding urinary sugar analysis



showed an equally curious result. Before glucose the urine contained 8.3% of sugar; at one hour it contained 7%, and at two hours 8%. Again the pre-glucose reading was the highest obtained.

Assuming that the first blood sugar estimation was correct (and the urinary sugar result suggests strongly that it was) the curious nature of the glucose curve is difficult to explain. It seems to me not unlikely that in this case the storage mechanism for sugar was extremely defective, and that when glucose was given the concentration of sugar in the blood was already such as to prevent any further absorption. Thus the glucose at the test meal would remain untouched, and only later would a certain amount be absorbed. This is shown by an increase in the urinary sugar at two hours, although the corresponding blood sugar reading was lower than any previous one. Storage of glucose must be continuing steadily, but probably now the glucose has passed beyond that part of the alimentary tract whence it can be absorbed with sufficient speed to maintain the blood sugar level.

Be these theoretical considerations as they may they cannot alter the classification of this case - a diabetic - either severe or on entirely erroneous lines of treatment. The subsequent progress of this case will be given in a later section when it will be clear that he was in reality a comparatively mild type.

Mrs. W. aged 56, a Housewife, was sent to me by her doctor for a glucose test. She had had sugar in the urine since the birth of her last child fifteen years before, and she had been dieted fairly strictly during all the intervening period. Recently she had had to have artificial dentures and, as she experienced some difficulty with these, she was most anxious to return to soft carbohydrate foods. The fact that she had remained fit for so many years made her regard such a suggestion as very reasonable.

She was a stoutly-built, healthy looking woman. I did not examine her but I was informed that the organs were sound. The family history was negative.

The result of the glucose test is shown on chart VI. No. 11, and it requires little comment. The initial blood sugar reading was high, and the return to the original level was defective. The urine contained much sugar both before and two hours after glucose. Clearly she was a diabetic, and no suggestion of a return to normal diet could be entertained.

ALLEN TREATMENT.

## I.

In 1913 Allen<sup>72</sup> in America conducted a series of experiments on the effect of extirpation of the pancreas in dogs. Diabetes was known to occur naturally in dogs, but rarely. Prior to this date a rapidly fatal diabetes had frequently been produced in these animals by complete removal of the pancreas. The successful production of a milder form of the disease by partial removal of the gland had, however, never been achieved, and it was to this problem that Allen addressed himself, and which he finally solved. He found that it was necessary to remove small portions of the gland in successive operations, and that by this means he could produce a diabetes of varying severity. Speaking generally a mild diabetes was produced when all but an eighth of the pancreas was removed; while if the remnant was less than a ninth of the whole gland a severe form of the disease resulted.

Having thus produced the disease experimentally Allen proceeded to try various forms of treatment. The methods in vogue, and the numerous 'cures' were tried without success; but in the course of his investigations Allen noticed that those dogs which were well fed died rapidly, while those which were poorly fed lived for a considerable time. Proceeding from this observation he evolved a pronouncedly undernutrition dietary upon which dogs rendered diabetic

could be kept alive indefinitely. The dogs lost weight, but the urine became sugar-free and thirst and polyuria disappeared. Thus thin and underfed they lived. On any form of liberal diet death rapidly supervened.

Not only did Allen prove the value of undernutrition diets in dogs; he did more than that. By numerous experiments he demonstrated that it was not the restriction or omission of any particular article in the diet which led to successful treatment. A diet of high carbohydrate value with low protein and fat was almost as disastrous as one of low carbohydrate and high protein and fat values. To ensure success in treatment it was necessary that the diet should be properly balanced, which meant that carbohydrate, protein and fat had to be present in certain proportions, the limits to which he discovered by experiment. This was an extremely important discovery, for from it sprang the modern system of dieting which revolutionised the treatment of diabetes, and placed it upon a sound scientific basis.

Following upon these successful experiments with animals the treatment by starvation and prolonged undernutrition was tested upon diabetic patients under the charge of Joslin in Boston, and of Stillman at the Rockefeller Institute. Soon it became clear that this mode of treatment was equally suited to the human diabetic, and it speedily superseded all other methods throughout the English-speaking countries. With increasing experience of its use certain slight modifications were introduced,

notably by Joslin; but the credit of originating the regime belongs to Allen, and thus I have called this type of treatment by his name.

It must be remembered, however, when considering the application of this treatment to diabetic patients that the cases of the partially depancreatized dog and the human subject are not strictly comparable. In the dog, with the employment of satisfactory technique the part of the pancreas which is left in situ is healthy, and probably remains healthy. In the human diabetic, on the other hand, the pancreas is diseased, and it is impossible, in the present state of our knowledge, to say whether we are dealing in any particular case with a progressive lesion.<sup>43, 51, 66.</sup> Be that as it may experience has proved the value of the Allen Treatment in the great majority of diabetics, the results obtained everywhere by its use being incomparably better than those recorded on any other type of treatment. I mention this point merely by way of explanation, because, as will be clear from the results given in this section, there were some cases which responded poorly to the starvation treatment, or which responded at first, but failed to react later. It may be, as Cammidge<sup>12</sup> believes, that it is those cases in which the pancreas alone is damaged which respond well to Allen treatment, while an unsatisfactory result on this treatment may mean that some other organ or organs are affected. At present the part played by the liver is receiving much attention,<sup>1, 7, 70.</sup> but much more work on the pathology of diabetes is required before we can come

to any definite conclusion on this matter. A discussion upon all the possible factors, however, lies without the scope of this paper.

As so frequently happens, about the same time as Allen was conducting his experiments, other workers had come to adopt a somewhat similar plan of treatment. Special mention must be made of Graham working in London. In 1915, while investigating the problem of ketosis with Hurtley, Graham found that the excretion of acetone bodies was much reduced by giving to diabetic patients a vegetable diet with eggs, a form of treatment advocated by van Noorden. He noted further that, if the diet of the patient was increased, acetone bodies returned to the urine; while glycosuria, which had also been reduced or cleared by the same treatment, again became very marked. Working from these findings Graham proceeded to treat his patients by greatly lengthened periods of undernutrition diet, any advance in the quantity of food given being very gradual. In this way he evolved his well-known "ladder diet". Further experimentation with this diet was interrupted by the War, and in consequence the details of his scheme of treatment were not published until <sup>1921</sup>~~1918~~<sup>79</sup>. Thus the credit of introducing the "undernutrition" treatment of diabetes must go to Allen.

It was not until March, 1920 that I had an opportunity of treating cases of diabetes on the lines laid down by Allen. In the Army in France cases of diabetes were rare, and such

cases as did occur were rapidly transferred to England, so that opportunities for treating them did not arise. During occasional periods of leave, however, I saw several cases in the Glasgow Royal Infirmary, and in 1918 Dr. Jack showed me two cases in his wards which were being treated with a completely vegetable diet devised by Williamson of Manchester.<sup>69.</sup> The results in these cases as regards the rapid disappearance of sugar and acetone from the urine were very striking, and greatly impressed me. Circumstances prevented my having an opportunity of trying this form of treatment myself for the following eighteen months, but in March 1920 through the kindness of Dr. Cowan I was permitted to supervise the treatment of the diabetic cases in his Wards, particularly with a view to instituting their routine treatment on the lines laid down by Allen, the details of which those of us who had been on service now learned for the first time.

The methods adopted by Allen and his co-workers were first carefully studied. Allen's "Monographs" gave an excellent description of his treatment, but the publications of Joslin were found to be most helpful from a practical standpoint. Needless to state it was not possible to follow out the regime there described in every respect. The medical staff was limited - all laboratory experiments had to be performed in the test-room attached to the wards: the nursing staff was constantly changing, and it was not found possible to obtain nurses for the treatment of diabetic patients only.



Throughout the treatment of the cases described in this section only the two Sisters in the wards were really familiar with the methods adopted, and I have to express my gratitude to them for very great assistance, particularly in the drawing up of diets, and in the keeping of charts. From the outset I endeavoured to render the treatment as simple and straightforward as possible, not only that it might be carried out in hospital under these handicaps, but also that it might readily be understood by the average patient, and so carried on after dismissal. For treatment of cases in Nursing Homes and in their own homes simplicity was also essential. At the same time it was recognised that in order to treat safely certain routine tests were necessary. At that time (as even to-day with insulin at our command) in diabetes of any severity acidosis was always a menace under any form of treatment, and it had to be discovered what tests were necessary, what useful, and what of no value. I shall describe later the routine ultimately adopted as regards these tests; but first I shall deal with the routine treatment of a diabetic patient of average severity on admission to hospital.

The three indications in any scheme of treatment of diabetes are as follows :-

- (1) To guard against acidosis.
- (2) To diet the patient.
- (3) To teach the patient to diet himself.

It must be clearly recognised at the outset that the

treatment of acidosis takes precedence over everything else. It is of vital importance that every effort be made to combat this symptom without regard for urinary sugar or any complication, save perhaps cardiac failure, with which it is so frequently associated. It is convenient, however, to describe first the routine treatment of the average case, although here again some qualification is necessary. Routine treatment of diabetes is bad treatment. Every case has to be considered on its merits, and no two cases can be treated in exactly the same way. The only sound method is to train the patient to treat himself - to regulate his own diet, test his own urine, and keep a weekly record of his weight. But obviously this was an unattainable ideal in hospital practice. Time was a consideration with almost every patient, and many were not sufficiently intelligent to learn. Moreover, if many patients were to be treated at one time some routine method was essential to allow the available nursing staff to overtake the work. For these reasons the following routine was adopted.

On admission the patient was weighed stripped, and put to bed. A full history of the case was made, family history and record of previous illnesses being especially enquired into. The date of onset of the condition was carefully sought for so far as such symptoms as thirst, polyuria, loss of weight, weakness, and glycosuria could determine it. The history of presence or absence of skin,

eye, or other complications was also noted. A complete physical examination was then made, particular attention being directed toward the state of the tongue and breath, the condition of the lungs, and the presence or absence of the knee jerks. The urine was tested at once for sugar, acetone, diacetic acid and albumen, and the collection of the 24 hours' output was commenced. An estimation of the blood sugar was made as soon as possible.

(For convenience the following contractions are used in the text:-

C - Carbohydrate.  
P - Protein.  
F - Fat.  
Cal- Calories.)

If the breath did not smell of acetone and the urine showed at most a slight reaction to the test for acetone, but none to the test for diacetic acid, the patient was put upon a diet consisting of C.100 P.100 F.110 (Sheet A. No. 1.) As is apparent this diet represented an average day's allowance so far as P. and F. were concerned, whereas the C. was about a quarter of the normal consumpt. The urine was collected from 7 a.m. to 7 a.m., the total amount was measured, and the amount of sugar passed in the 24 hours was calculated and charted. In the morning a fresh specimen of urine was obtained and examined for acetone and diacetic acid. All being satisfactory the patient's diet was changed on the second day to C.60 P.25 F.5. Here the C. was slightly reduced, but P. and F., especially F., were greatly curtailed. The reason for retaining a certain amount of C. in the diet was to prevent the development of an acidosis, the C.

serving, so it was believed, to assist in the katabolism of any surplus F. which might be present in the body. In this I followed the routine practice which Joslin had advised in the early treatment of all cases of diabetes. By this means death from a starvation acidosis (an exaggeration of the acidosis which normally occurs in fasting, and an occasional occurrence under the Allen routine of commencing starvation on the day of admission) was rendered much less probable; while the slight loss of time which this procedure involved was more than compensated by the increased safety. How our views on this matter were later modified as the result of the experiments of Newburgh and Marsh<sup>50</sup> I shall explain below.

On the third day the patient was given C.30 P.5 F.O. F. was now cut completely out of the diet, and a small amount of C. was retained. On the fourth day, all being well - the patient's general condition satisfactory, and no evidence of severe acidosis - the patient was placed on full starvation dietary. On this day fluids were permitted ad lib. - water, lemon drinks, tea, and coffee. Thin meat soups with no food value, such as Oxo and Bovril, were also allowed. If it appeared wise a moderate amount of alcohol in the form of whiskey or brandy was added. In the majority of cases the urine passed during the first full starve day was sugar-free; but if glycosuria persisted starvation was continued for a further 24 hours. On the fifth day if sugar had disappeared from the urine on the first day of starvation, or on the sixth day if a

NORMAL CASE WITHOUT ACIDOSIS.

Table I

	Carbohydrate.	Protein.	Fat.	Calories.
1.	100	100	110	1790.
2.	60	25	5	385.
3.	30	10	0	160.
4.	10	0	0	40.
5.	0	0	0	0.

Starve till sugar-free or for three days.

6.	10	0	0	40.
7.	20	5	0	100.
8.	30	10	0	160.
9.	40	15	5	265.
10.	50	20	5	325.
11.	60	20	10	410.
12.	70	30	15	535.

13. Starve.

14. Add 10 g. C. until sugar appears in the urine,  
each day.

15.	Tolerance - 20 g.	40	25
16.	"	50	30
17.	"	60	35
18.	"	60	45
19.	"	80	55

20. " Add P. until 1.5 g. per kilo weight.

21. Starve.

22.	"	"	70
23.	"	"	85
24.	"	"	100
25.	"	"	110

second starve day had been necessary, and whether or not the urine was sugar-free, feeding was resumed with C.10 P.O F.O, and thereafter the diet was increased by 10 gms. C. per day. If, however, sugar had not disappeared from the urine after the second day's starve the patient was given two days on C.10 P.O F.O and then starved for a further 24 hours. Sugar having now disappeared from the urine (the treatment of the exceptional cases will be dealt with later) the diet was increased daily as in those whose urine had become sugar-free on a 24 or 48 hours starve. The addition of 10 gms. of C. was continued daily, as shown in Table I, until sugar re-appeared in the urine. This was the signal for another starve day, which, in practically every case, cleared up the glycosuria once again. Next day feeding was resumed, but the amount of C. was reduced to 10-15 gms. below the level at which glycosuria had occurred. This level at which sugar re-appeared in the urine was called the "C. tolerance level".

Broadly speaking P. was added to the diet in a manner similar to the addition of C. Allen had advocated the giving of C. alone at first until the C. tolerance was found; then the addition of P. gradually until the patient was receiving 1-1.5 gms. per kilo of body weight. In these diets P. was given much earlier than Allen recommended, the patient receiving 5 gms. of P. on the second day of diet and thereafter a small increase of 5 gms. almost daily (see Table I) while the C. tolerance was being found. When the tolerance

had been found P. was increased by 10 gms. daily until the patient was receiving the requisite 1.5 gms. per kilo of body weight. It is admitted that this vitiated to a slight degree the purity of the C. tolerance test, but the gain in time was considerable, and this was an important matter, particularly in hospital cases. In addition the diets were rendered more palatable, a circumstance which reduced the likelihood of unauthorised food being eaten as was always liable to happen in children, and in the less intelligent of infirm patients. To the more modern practice of maintaining the P. content of the diet throughout I shall refer later.

The diet having been advanced to this stage Allen recommended that F. be added until the patient ceased to lose weight. Again, as will be clear from the diets given in Table I, I gave F. fairly early also, but always very cautiously, and controlling each advance by careful testing for evidence of acidosis. When, in the average adult, the weight was being maintained, no further increase was made in the fat allowance.

The carrying out of the process described above whereby an "equilibrium" diet was arrived at usually occupied from 21 to 28 days. A reference to Table I will show that every seventh day was a "starve day". This interpolating of a weekly starve day was recommended by Allen, and, in my experience, was extremely useful. Not only did it tend to remove any excess of sugar from the blood and so to raise the food tolerance, but it served the equally important purpose of initiating patients

into this routine. It was almost invariably found that the suggestion of a day's starvation was extremely distressing to most patients, but the actual experience proved their apprehensions to have been groundless. Many even stated that they always felt better after such a day, and in no case was difficulty experienced in having this routine carried out.

As a result of following this plan of dieting the patient should be free from glycosuria throughout the 24 hours on a diet of (1) Carbohydrate - 10 gms. below the C. tolerance: (2) Protein - 1-1.5 gms. per kilo of body weight: (3) Fat - to maintain weight. The question naturally arises, what is the optimum weight? Obviously this must vary very greatly as it does in normal healthy adults; but it is of primary importance that the weight be kept low. Speaking generally I found that a fairly satisfactory guide was the minimum weight for height as obtained from insurance tables; but very many patients, including all the severe cases, carried on at weights much beneath this standard. More recently somewhat complicated forms (an example of which is appended) called nomographs have been published, from which it is possible to calculate the amounts of C.P. and F. requisite in a patient of known weight, and also the number of calories required. I shall return to this point later.

It will be clear from what I have stated above that the patient was carefully observed throughout this period of treatment. The main points may be summarised as follows :-



DAILY: Morning and evening Temp. Pulse. Resp.

Morning blood-sugar estimation.

Morning specimen of urine for Sp. Gr.,  
Reaction, Acetone, Diacetic Acid.

Collection of 24 hours output of urine for  
quantity, and amount of sugar if present.

State of breath, tongue and bowels.

BI-WEEKLY: Thorough physical examination.

Weight.

### DIETS.

The standard diets which I employed are shown in the accompanying table. These diets were arrived at after extensive trial, and were built up with a view to simplicity and availability of materials. Such diets were employed in the initial stages of all cases, mild or severe, save where there was evidence of considerable acidosis. Even in cases showing marked emaciation the same procedure was followed as has been described above.

Enclosure A. gives in detail the constituents of the diets listed above. Each diet represents the amount of food to be taken in 24 hours, the various quantities being split into meals as required. The only stipulation made was that the C. of the diet must be divided over the day, so that no large amount was taken at any one time, and in this way it was hoped to prevent the 'flooding' of the circulation with sugar as must follow the ingestion of any large amount of C. at one meal.

As will be apparent an endeavour was made to make the meals approximate as closely as possible to the average type. Thus, where allowed, bacon and egg were allocated to breakfast; soup and meat to dinner; and so on. By this means it was hoped to get rid to some extent of the feeling of being 'different' from everyone else, and, by utilising the foodstuffs such as healthy members of the family might take, to make it more probable that the poorer patient would keep to his diet. For a like reason only common articles of diet were included in these lists; but where circumstances and intelligence permitted other foodstuffs could be substituted as I shall explain later.

The food values given in the above series of diets were derived from the following sources :-

- |   |            |
|---|------------|
| (1) Food and Principles of Dietetics.   | Hutchison. |
| (2) Diabetic Cookery.   | Cambridge. |
| (3) Diabetic Manual.  | Joslin.    |
| (4) Diabetes.   | Horowitz.  |
| (5) The Food Supply of the United Kingdom. Appendix I B. 1916.<br>Report by Committee of Royal Society. |            |

Small differences were found in the case of almost every food, either in the estimated C.P. or F., or even in all three. In each an average was struck, and the value fixed in gms. for each constituent. In dealing with P. and F. I considered it sufficiently accurate to disregard smaller fractions, and to reckon to the nearest 5 gms. Thus 23 gms. was reckoned as 25 gms.:

22 gms. as 20 gms. The smaller value C. foods I dealt with in a similar manner, but with foodstuffs of higher C. content greater care was requisite, and the calculation was made to the nearest gm. Later, when it became clear that the P. content of the diet was of great importance also, I revised the P. values and altered some of these to make them more accurate. Thus the P. in white bread was altered from 10 gms. to 12 gms., and that of green peas from 0 to 2.5 gms.

Beside each diet is stated its Calorie value. In calculating this value 1 gm. of C. was reckoned as yielding 4 Calories: 1 gm. of P. 4 Calories: 1 gm. of F. 9 Calories.

This represents the routine treatment of the average patient who showed on admission no pronounced evidence of acidosis. As will be clear from an examination of the case records many alterations were necessary in individual cases, but the treatment of three groups may be considered here.

(1) Children: These cases were treated in bed throughout almost their entire period of observation. It was found impossible otherwise to control their diets, and this was so important a matter that the advantages of exercise had to be sacrificed. In stronger children, of course, a return of sugar to the urine could always be followed by a starve day, and so disobedience could be punished; but in the weaker children this could not be resorted to frequently. As regards diet, the

amounts of P. had to be relatively much greater than in the adult to allow of normal growth. 1.5 gms. per kilo of body weight was never found to be sufficient. 2-3 gms. per kilo were always necessary. <sup>24.</sup> F., too, had to be given in increased amount. I found it a safe procedure to give as much F. as could be tolerated without giving rise to acidosis or to gastro-intestinal disturbances.

Obese Patients: In dieting stout patients rather more care had to be exercised during the period preceding the first starve day, while the diet was being reduced. With so much readily available fat in his own tissues the patient was liable to develop a very considerable acidosis on diets such as those given above; but a more gradual reduction, with an earlier cut in the F. and a more generous C. ration, generally prevented such an occurrence.

With regard to weight, it was of fundamental importance that a marked reduction should be made in these cases, and, by means of prolonged undernutrition dietary, such a result was not difficult to achieve. Needless to say, during this period patients frequently complained of weakness and of inability to work: but for successful treatment such a regime was essential. When weight had been reduced to what was

considered satisfactory then diet was advanced as in the average case to allow 30 Calories per kilo of body weight or as near to that as tolerance would permit.

Emaciated Patients: These cases were the most difficult to deal with, mainly because the disease in them was of a very severe character or of long standing. The earlier stages of treatment were similar to those adopted in the average case as described above, but every endeavour was made to reduce the period of starvation to a minimum. Generally two or even three brief periods of starvation (48 hours) were necessary before the urine was rendered sugar-free, these periods alternating with periods of light feeding. In the majority of patients sugar was banished from the urine in this way, but in a few cases it was found impossible to achieve this result. In the latter severe undernutrition had to be abandoned after a time lest death ensue from inanition, and, with increasing feeding, one had to be satisfied with keeping the urinary sugar at a minimum. On the whole it was surprising how few cases proved to be so refractory, the great majority responding readily to the starvation dietary. In these cases alcohol was used more freely to maintain strength, with caffeine and digitalis where cardiac weakness was apparent. Again it was rather surprising how seldom these

adjuvants were required.

As in the average case an endeavour was made to bring these patients up to a reasonable weight, and ultimately to allow a dietary providing 25-30 Calories per kilo of body weight. But frequently the C. tolerance was found to be extremely low, perhaps under 20 gms. in the 24 hours, and to provide the necessary Calories the amounts of P. and F. had to be correspondingly high.

This in turn led to the development of acidosis, the bugbear of the older dietetic methods, to which this was virtually a return. A compromise thus became necessary, and, as the result of experience, I found it best to increase the C. to such an extent as to allow P. and F. being added in sufficient quantity to provide 20 Calories per kilo of body weight without causing acetone to appear in the urine. On such a diet sugar appeared in the urine, but never in great amount. While recognised as highly undesirable one had to be content with merely keeping such sugar in check as the best practical solution of the difficulty at that time. Of course, these patients were fitted for the lightest forms of work only; but as the alternative was death either from inanition or in coma the choice was not difficult to make.

Such, then, was the routine procedure employed both in hospital and in private practice. During the initial stages of investigation and dieting every facility was given to the patient to understand and to appreciate all that was being done. The necessity for meticulous care in dieting, and the importance of keeping the urine sugar-free were carefully explained: and above all it was impressed upon him that success was impossible unless the treatment was carried out with sedulous particularity after dismissal. The regular weighing of all food was particularly emphasised. The patient was provided with a readable book, such as Joslin's Diabetic Manual, and with its aid he was taught the principles of treatment as outlined above so far as he was capable of learning. The method of building up the diet was also carefully explained, and, given a large table of values, it was obvious to each that a very big variety in the meals was possible. To the poorer patients, however, so large a variety was not to be procured, while for the less intelligent it was essential to make the method as simple and readily understood as possible. With this end in view I compiled the following list of foodstuffs with their corresponding values :-

	<u>Carbohydrate.</u>	<u>Protein.</u>	<u>Fat.</u>
White Bread - toasted	60	12	0
"        "	55	8	0
Diabetic Bread	0	30	20
Oatmeal	70	16	8

	<u>Carbohydrate.</u>	<u>Protein.</u>	<u>Fat.</u>
Porridge	20	2	0
Milk	5	3	4
Cheese	3	25	30
Cream	4	3	30
Butter - Margarine	0	0	85
Ham	0	20	35
" - boiled	0	20	25
Bacon	0	10	60
Egg	0	6	6
White Fish	0	20	0
Chicken	0	25	5
Mutton	0	25	20
Beef	0	25	20
Gelatine	0	90	0
Horlick	67	16	9
Green Peas - fresh	15	5	0
Rabbit - roast	0	40	10

### Vegetables

- 5%     Lettuce, Cucumber, Spinach, Rhubarb, Asparagus, Marrow, Celery, Mushrooms, Tomatoes, Brussel Sprouts, Watercress, Seakale, Cauliflower, Cabbage, Leeks, Radish, Grapefruit, Oysters.
- 10%    Stringbeans, Pumpkins, Turnips, Beets, Carrots, Onions, Melons, Strawberries, Peaches, Lemons, Gooseberries, Oranges, Brazil Nuts.
- 15%    Artichokes, Parsnips, Raspberries, Currants, Apricots, Pears, Apples, Cherries, Walnuts.



Vegetables:

20% Potatoes, Beans, Boiled Rice, Macaroni, Plums,  
Bananas.

When cooked the 5% group count as 3%  
" " 10% " " 6%.

30 gms. - 1 oz.

This contained the bulk of the commoner articles of diet available to hospital patients. Almost without exception values are a little high in order that any error on the part of the patient, or any tendency to increase the food ration might, to some extent, be compensated.

When the stage of investigation was completed and the diet fixed the subsequent treatment followed one of two courses.

(1) If the patient did not seem to be sufficiently intelligent to test his own urine, build up diets, and otherwise to carry out treatment for himself, a reversion had to be made to the old method of supplying him with a copy of the diet finally fixed upon, and instructing him to continue on that diet. Invariably he was given two or three variations, and in each the gramme weights were converted into ounces as being a more familiar weight. The patient was instructed to return once a week to report progress, bringing with him a sample from the urine passed during the previous 24 hours. In the earlier days the more severe cases returned to hospital for a week-end, when their condition was thoroughly investigated

and the necessary adjustments in diet were made; but as patients became more numerous it was found impossible to find accomodation for them, while those who had found work could not afford the necessary time. A Sunday visit had to suffice in these cases, but if they broke down they were admitted for a further period of treatment.

This method was obviously unsatisfactory. After a time patients became tired of such limited feeding, and broke diet; but experience showed that this method was to be preferred to allowing a patient to build diets with no proper understanding of what he was doing. Unfortunately the majority of hospital patients, especially the women, had to be treated on these lines. A weekly starve day (generally Sunday) was insisted upon in all these cases, and this proved extremely helpful in checking glycosuria.

- (2) The intelligent patient presented little difficulty as regards after-treatment. On leaving hospital he could build up diets with ease, and split them into meals: keep a record of his weight: and test hiw own urine for sugar. Some patients could also test for acetone. Such a patient reported weekly for a short period, and thereafter monthly, or whenever the re-appearance of sugar or acetone caused anxiety. Diet was varied according as various foods came into season, and thus a very large variety was possible. A few infirmiry cases, and most private cases, either carried on such treatment themselves or had it done for them by a relative or nurse. Where,

as occasionally happened, the patient refused to exercise the necessary care as being too much trouble, he had perforce to be allowed to get along as best he could. Invariably a return of symptoms brought such patients back, and, having learned their lesson, they seldom broke diet again.

Acidosis: As Naunyn had pointed out many years before acidosis was the chief danger in all cases of severe diabetes. The case which was admitted smelling strongly of acetone and with a strong Tinct. Ferri Perchlor. reaction in the urine, or with air hunger, or in coma, was readily recognised as demanding urgent and vigorous measures of treatment; but difficulty was frequently experienced in deciding which case could safely be starved and which required a preliminary course of 'acidosis dietary'. The tests which I then employed in addition to the urinary tests were

- (1) The Estimation of the Alveolar  $\text{CO}_2$ .<sup>75</sup>
- (2) The Ammonio -Nitrogen Ratio, as obtained from the urine.<sup>38</sup>

(1) The normal  $\text{CO}_2$  content of the alveolar air is about 5%. A reading under 4% denoted a severe acidosis, with the likelihood of coma supervening. Readings around 2% denoted impending coma. The apparatus I employed was that of Fredericia, a simple and readily portable form. But a sample of alveolar air is notoriously difficult to secure, and the results obtained were seldom conclusive save in those cases which the ordinary clinical examination showed to require cautious treatment.

(2) The urinary Ammonio -Nitrogen ratio is normally 1 : 20 . Readings below 1:10 excite suspicion: readings around 1:5 denote a serious condition. Results obtained from a single specimen of urine were never found to be reliable, while to wait for a specimen of the 24 hours collection was obviously very undesirable. The results, too, were much influenced by the character of the food consumed, and also by any previous drugging with alkali, as had so frequently been prescribed. Thus this method also was not very helpful, and, at most, served only as a corroborating factor in the general conclusion.

Experience proved that the only safe method of treatment was to consider every doubtful case as likely to develop coma, and to treat it accordingly. The following case occurring early in my series illustrates how deceptive such patients may be.

W.J. age 17, was admitted to hospital on 22:11:20 complaining of thirst and frequency of micturition of five weeks' duration. He had lost weight rapidly during that time.

He was a youth of moderate development, but poorly nourished. Physical examination was negative, save that the knee jerks, while present, were elicited with difficulty. He looked rather unwell, but he was bright and alert and made no complaint. On the night of admission he slept well, and in the morning seemed as well as on the previous day. The

tongue was cleaner though the bowels had not acted. The urine was loaded with sugar; acetone was present, but there was no diacetic acid, and no odour of acetone in the breath. A fairly liberal carbohydrate diet was ordered (Table II.1) but during the day he complained of headache and of difficulty in swallowing his food, and in consequence he ate little (about 30:36:50) during the day, although he drank copiously. In the evening he became drowsy, and later on very restless, and he remained in this condition till 3 a.m. on the following morning. He then suddenly collapsed, the pulse becoming almost imperceptible; but on stimulation he revived, and at 8 a.m. he was much better. At that time his breath had a slight odour of acetone, and although the acetone in the urine was ++, the reaction to Tinct. Ferri Perchlor. was negative. By 11.30 a.m. his pulse had improved very perceptibly, and he was quite clear and intelligent. He was now on Oatmeal Diet. About 1 p.m. he commenced to become drowsy, and at 2 p.m. he could not be roused. He died at 3 p.m.. At no time was there any dyspnoea or air hunger, the breathing throughout being easy, regular, and natural.

The effect of such a case was to convince me, as I have stated above, that every doubtful case must be treated as though coma were imminent. The first essential in all cases is to get rid of the acidosis, or to reduce it to such an extent as to render starvation treatment safe. I employed for this purpose an oatmeal diet, giving small quantities

frequently, together with as much fluid in the form of water, soda water, tea, and soup as the patient could take. The oatmeal diet was really a modification of that recommended by van Noorden, and was prepared by soaking 300 gms. of oatmeal in 3000 c.cs. of water, straining, then boiling for ten minutes. 300 c.cs. of the resulting gruel were given 2 hourly. The value of this diet was approximately C.60: P.12: F.12. No eggs or butter were included as in van Noorden's regime as tending to increase the acidosis.

Sod. Bicarb. was given in large doses despite Allen's condemnation of this drug.<sup>3, 4</sup> Sometimes it appeared to be beneficial, and I was satisfied that in no case did it do any harm.<sup>5, 7, 8</sup> Intravenous Sod. Bicarb. was given on three occasions. In two of these the patient was in coma when admitted, and died shortly afterwards. In the third case (No. 1) the injection was given on the first sign of air hunger. In 15 minutes the air hunger passed off, and the patient made an excellent recovery from his threatened coma.

In the earlier cases, if the acidosis were distinct but less marked than to require the 'oatmeal treatment' described above, another series of diets was employed.

These diets differed from those in Table I in two particular respects (1) the carbohydrate was greater in amount, and its withdrawal from the diet was more slowly accomplished: (2) the amount of fat was relatively small. A brief experience proved, however, that such cases were unsuited to any routine

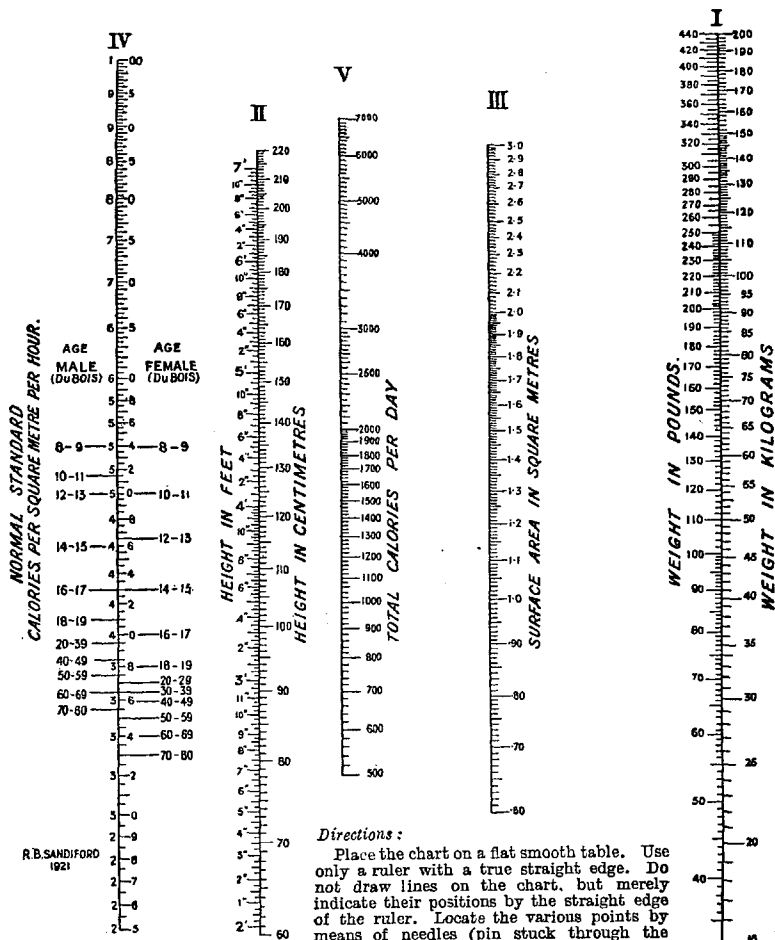
treatment, the diet being liable to alteration from day to day - according to the progress of the case.

It was found, too, that cases without exception could very safely commence treatment on the second diet in Table I (60:25:5), and latterly the first diet (100:100:110) was omitted, except in the very mildest cases. The same diet (No. 2) was found equally suitable for cases which had required a preliminary 'oatmeal period', such cases passing on from that diet to the usual routine. Thus the special 'acidosis routine' was dropped as being unnecessary.

These forms of treatment represent the methods employed from 1920 to 1922. There then came under my notice the work of Newburgh and Marsh,<sup>50</sup> of Woodyat,<sup>71</sup> and of Shaffer.<sup>76</sup> This work was mainly concerned with preventing the formation of acetone bodies by means of properly balanced diets, and with the amounts of protein required by the individual. Schaffer calculated the amounts of fatty acid which could be formed from P. & F. - the ketogenic substances: and also the amounts of glucose - the antiketogenic substances - which could be formed from C.P. and F.

$$\frac{\text{(The sum of the ketogenic substances)}}{\text{(The sum of the antiketogenic substances)}}$$
 he called the Aketogenic Ratio, and this value he put at 1.5. The calculation was made possible by the discovery of the following facts :-

# DIABETIC DIET CHART I



Large scale nomographs may be obtained from W. B. Saunders Co., West Washington Square.

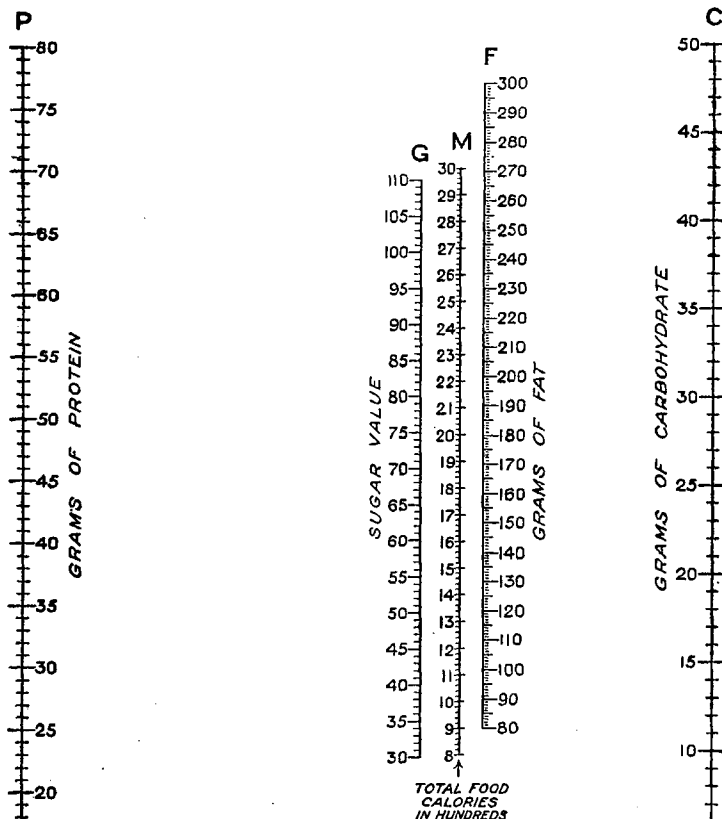
Extracted from

Graham's

THE PATHOLOGY AND TREATMENT OF DIABETES MELLITUS.



## DIABETIC DIET CHART II



### Directions:

#### (1) FOR BASAL MAINTENANCE DIET. (1:65 to 1 ratio.)

Determine basal maintenance calorie requirement (total basal calories per day) for the proper height and weight, age and sex of the patient on Chart I as per directions given there. On Chart II locate the calories thus determined (scale M), locate the grammes of protein on scale P. Between  $\frac{1}{2}$  of a gramme and 1 gramme of protein per kilogram of body weight are desirable. A ruler connecting these two points gives the grammes of carbohydrate and the grammes of fat at the points on scales C and F, respectively, where it crosses them. At the intersection with scale G it gives the sugar value of the diet in grammes. The food mixture thus estimated has a ketogenic-antiketogenic ratio of 1:65 to 1.

#### (2) FOR MAXIMUM CALORIE DIET (INSULIN).

Having determined on scale G the sugar value of the basal maintenance diet, add the number of grammes of glucose to be balanced by insulin. Locate this value on scale G. Any straight line intersecting scale G at this point and crossing the other scales will give a diet that has a ketogenic-antiketogenic ratio of 1:65 to 1.

Philadelphia, U.S.A.; or from H. N. Elmer, 1641, Monadnock Buildings, Chicago, U.S.A.

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100 gms. Carbohydrate give 100 gms. Glucose, 0 gms. Fatty Acid.

100 gms. Protein " 58 gms. " ,46 gms. " "

100 gms. Fat " 10 gms. " ,90 gms. " "

Accepting these values Woodyat devised the following formula:  $F = 2 C + 0.54 P$ . or as now generally written

$F = 2 C + \frac{1}{2} P$ ., which meant that in order to avoid the production of acetone in the urine due to unbalanced diet, the F. of the diet must not exceed twice the C. plus  $\frac{1}{2}$  the P. The formula was called the Woodyat Formula.

To use this formula the calorie value of the diet was first decided upon, and the most convenient method of fixing this figure was to find the number of calories required for basal metabolism. This is readily found by using either prepared tables such as those of du Bois,<sup>78</sup> or the nomograph of Boothby and Sandiford, a copy of which I include here. The amount of P. required was first fixed. I have stated above that 1-1.5 gms. per kilo of body weight was generally reckoned as being the optimum amount; but Newburgh and Marsh showed by experiment that smaller quantities - 0.6 gms. per kilo of body weight - were quite sufficient for the diabetic patient's needs, and, as almost half the P. given went to form sugar, they strongly recommended that a minimum of P. be prescribed. This figure then having been fixed, the amount of C. and F. were easily calculated from such a nomograph as that of Wilder<sup>78</sup> (see reverse of Boothby and Sandiford nomograph.) A diet so calculated is based on an aketogenic ratio of 1.6.

Woodyat also pointed out that when a patient ~~is~~ starved he utilises his own P. and F., and thus it would be sounder treatment to feed on diets calculated as above, and so to minimise the loss of weight. This suggestion found general acceptance in America, where prolonged periods of starvation dietary were abandoned and replaced by diets low in P. but comparatively rich in C. and F. The patient was given the appropriate diet, and allowed to continue unchanged until he ceased to pass sugar in the urine. Acidosis, it was argued, could not arise through starvation on such a diet, nor from the food provided as the calculated aketogenic ratio proved.

In view of these results I scrutinised carefully the diets which I had employed, and which have been given in detail above. In all the aketogenic ratio was satisfactory: indeed in the majority the aketogenic factors were in generous proportion. The P. ration, however, was greater than that recommended by Woodyat, but the diets generally were more in accord with his revised treatment than with the strict prolonged undernutrition diets of Allen. However, several cases were treated at this time on diets built up in strict accordance with Woodyat's recommendations, but in no case were the results so satisfactory or so rapidly achieved as under the old routine. Graham<sup>20</sup> records a similar finding, and suggests as an explanation that such feeding overlooks the importance of pancreatic rest to allow of the recovery of the  $\beta$  cells of the Islands of Langerhans.

Thus the scale of diets described above, arrived at through practical experience and found to give very satisfactory results, was subsequently proved to be even sounder practice than the rigid adherence to the Allen principles: while the advance in diet was sufficiently slow to prevent overstrain of the pancreatic cells. In these diets one would thus seem to find a satisfactory compromise.

Enclosure B. shows a few typical diets constructed upon the lines described above.

DIABETIC DIETS.

ENCLOSURE A.

# STANDARD DIABETIC DIETS.

1.	Carbohydrate.	Protein.	Fat.	Calories.		Gramme s.
	Grammes. 100	100	110	1790.	Cooked 5% Vegetables	200
					Fresh 5% "	190
					Potato	75
					White Bread	100
					Peas	100
					Beef	125.
					Ham	125.
					Fish White	95.
					Eggs	2
					Butter	31

2.	60	25	5	385.	Cooked 5% Vegetables	250
					Fresh 5% "	150
					Potato	50
					White Bread	50
					Peas	50
					Chicken	40
					Fish (white)	50
					Butter	5.

3.	30	10	0	160	Cooked 5% Vegetables	330
					Fresh 5% "	200
					Potato	50
					Fish (white)	50

4.	10	0	0	40	Cooked 5% Vegetables	180.
					Fresh 5% "	100.

5. Tea, Bovril, Coffee, ad lib.

Fast until sugar-free. Maximum fast three days.

6.	10	0	0	40.		
----	----	---	---	-----	--	--

2).

7.

20	5	0	100.	Cooked 5% Vegetables	200.
				Fresh 5% "	200.
				Potato	20.
				Fish (white)	25.

-----

8.

30	10	0	160.	Cooked 5% Vegetables	300.
				Fresh 5% "	200.
				Potato	55
				Fish (white)	50

-----

9.

40	15	5	265.	Cooked 5% Vegetables	250.
				Fresh 5% "	100.
				Potato	50.
				Peas	25.
				Fish (white)	60.
				Butter	6.
				White Bread	25.

-----

10.

50	20	5	325.	Cooked 5% Vegetables	160.
				Fresh 5% "	150.
				Potato	50
				Fish (white)	80
				Butter	6
				White Bread	50.

-----

11.

60	20	10	410.	Cooked 5% Vegetables	200.
				Fresh 5% "	100
				Potato	50
				Peas	50
				Onion	50
				Chicken	40
				Eggs 1	
				Butter	3
				White Bread	50.

-----

12.

Carbohydrate.	Protein.	Fat.	Calories.		Grammes.
Grammes. 70	30	15	535.	Cooked 5% Vegetables	200
				Fresh 5% "	100
				Potato	50
				Chicken	47.
				Eggs 2	
				Butter	3
				White Bread	80.

-----

13.

Starvation day.

14. Add grammes 10 carbohydrate each day until sugar appears in the urine.

-----

A.	TOLERANCE - 20	40	25
B.	"	50	30
C.	"	60	36
D.	"	70	45
E.	"	80	55
F.	Add protein, grammes 10 daily until it equals 1.5 gms. per kilo body weight.		

G.

Starvation Day.

-----

a.	Add fat to carbohydrate tolerance - 20, protein as required.		
b.	x	y	70
c.	x	y	85
			100

-----



Carbohydrate. Protein. Fat. Calories.

Grammes..

Grammes	70	30	15	535.	Cooked 5% Vegetables	200.
					Fresh 5% "	270.
					Potato	100
					Peas	25
					Fish (white)	65
					Eggs	2
					Butter	4
					Bread (white)	50

-----

	40	40	25	545.	Cooked 5% Vegetables	300
					Fresh 5% "	300
					Potato	50
					Onion	100
					Chicken	75
					Butter	12
					Eggs	2
					Fish (white)	50

-----

	50	50	30	670.	Cooked 5% Vegetables	230
					Fresh 5% "	200
					Potato	75
					Peas	25
					Chicken	115
					Eggs	2
					Butter	7
					Bread (white)	25.

-----

	40	60	35	715.	Cooked 5% Vegetables	300.
					Fresh 5% "	300
					Potato	75
					Mutton	100
					Fish (white)	145
					Eggs	1.
					Butter	10

-----

Carbohydrate.	Protein.	Fat.	Calories.	Grammes.
Grammes. 70	50	30	750.	Cooked 5% Vegetables. 200. Fresh 5% " 170 Potato 75. Beef 100 Fish (white) 65 Eggs. 1 Bread (White) 75 Butter 5.
-----				
80	60	35	875.	Cooked 5% Vegetables. 200. Fresh 5% " 170. Potato 100 Peas 25. Beef 100 Fish (white) 80. Eggs 2. Butter 4. Bread (White) 75.
-----				
90	70	45	1045.	Cooked 5% Vegetables. 100 Fresh 5% " 200 Potato 50 Onion 100 Orange 50 Beef 100 Ham 50 Fish (white) 105 Eggs. 1 Butter 4 Bread (white) 100
-----				
70	80	55	1095.	Cooked 5% Vegetables 200 Fresh 5% " 170 Potato 50 Peas 25 Beef 125 Ham 50 Fish (white) 100 Eggs. 2 Butter 3 Bread (white) 75.

ENCLOSURE 'B'

# DIABETIC DIETS.

Carbohydrate.	Protein.	Fat.	Calories.		Grammes.
Grammes. 60	25	5	385.	Cooked 5% Vegetables	300
				Fresh 5% "	200.
				Potato	70.
				Fish (white)	100
				Butter	6
				White Bread	50.
*****					
30	10	0	160.	Cooked 5% Vegetables	330.
				Fresh 5% "	300
				Orange	50
				Gelatin	11
*****					
20	5	0	100.	Cooked 5% Vegetables	200
				Fresh 5% "	200
				Onion	70
				Fish (white)	25.
*****					
40	15	5	265.	Cooked 5% Vegetables	300
				Fresh 5% "	120
				Potato	50
				Butter	4
				Chicken	50
				White Bread (toasted)	25
*****					
50	20	5	325.	Cooked 5% Vegetables	300
				Fresh 5% "	120
				Potato	100
				Chicken	50
				Fish (white)	25
				Butter	4
				White Bread (toasted)	25.
*****					
60	20	10	410.	Cooked 5% Vegetables	200.
				Fresh 5% "	150
				Potato	65.
				Orange	60
				Fish (white)	50
				Eggs. 1	
				Butter	5
				Bread (white)	50
*****					

Carbohydrate. Protein. Fat. Calories. Grammes.

Grammes. 50	70	45	885.	Cooked 5% Vegetables	250.
				Fresh 5% "	225.
				Potato	50.
				Peas	50
				Chicken	100
				Fish (white)	125.
				Ham	50
				Eggs 1	
				Butter	20
				Bread (white)	25.

---

40	80	55	975	Cooked 5% Vegetables	300.
				Fresh 5% "	220
				Potato	50
				Orange	100
				Gelatin	11
				Mutton	100
				Fish (white)	165.
				Eggs 2.	
				Butter	27.

---

40	80	70	1110	Cooked 5% Vegetables	200
				Fresh 5% "	200
				Potato	75
				Onion	100
				Peas	20
				Mutton	100
				Ham	50
				Fish (white)	160
				Eggs. 2	
				Butter	24.

---

80	40	25	705.	Cooked 5% Vegetables	300
				Fresh 5% "	200
				Potato	100
				Chicken	90
				Eggs 2.	
				Butter	12
				Bread (white)	75

Carbohydrate. Protein. Fat. Calories.

Grammes.

Grammes.	80	80	70	1270.	Cooked 5% Vegetables	200
					Fresh 5% "	160
					Potato	100
					Onion	85
					Beef	100
					Ham	50
					Fish (white)	135.
					Eggs. 2	
					Butter	25
					Bread (white)	75

---

	30	80	85	1205	Cooked 5% Vegetables	300
					Fresh 5% "	200
					Potato	60
					Chicken	100
					Ham	125.
					Fish (white)	90
					Eggs. 2	
					Butter	27

---

	40	90	100	1420.	Cooked 5% Vegetables	200
					Fresh 5% "	205
					Potato	50
					Beef	150
					Fish (white)	100
					Ham	100
					Eggs 2.	
					Butter	28
					Bread (white)	25

---

	50	100	110	1590.	Cooked 5% Vegetables	165
					Fresh 5% "	220
					Potato	50
					Orange	100
					Beef	150
					Ham	200
					Fish (white)	70
					Eggs 1.	
					Bread (white)	25
					Butter	5

Carbohydrate. Protein. Fat. Calories.

Grammes.

Grammes. 80	80	85	1405	Cooked 5% Vegetables	200
				Fresh 5% "	250
				Potato	50
				Onion	66
				Orange	100
				Beef	100
				Ham	100
				Fish (white)	90
				Eggs. 2	
				Butter	20
				Bread (white)	60

---

80	90	100	1580.	Cooked 5% Vegetables	200
				Fresh 5%	170
				Potato	50
				Fish (white)	70
				Orange	100
				Peas	50
				Mutton	100
				Ham	160
				Eggs. 2	
				Butter	15
				Bread (white)	75.

---

90	100	110	1750	Cooked 5% Vegetables	150
				Fresh 5%	150
				Potato	100
				Mutton	100
				Fish (white)	50
				Cheese	100
				Eggs. 2.	
				Butter	26
				Bread (white)	100
				Ham (boiled)	100

BENGERS FOOD VALUES.

	Carbohydrate.	Protein.	Fat.
DRY	80	12	1
Formula 1.	6	1.5	1
" 2.	7	1.5	2.5
" 3.	6	2	2
" 4.	9	3.5	3

	Benger.	Sugar of milk.	Water.	Milk.	Cream.
Formula 1.	$\frac{1}{4}$	$\frac{1}{3}$	8	4	ounces.
" 2.	$\frac{1}{2}$	$\frac{1}{5}$	8	3	1 cream.
" 3.	$\frac{1}{2}$		6	6	
" 4.	$\frac{3}{4}$		2	10.	

ALLENBURY DIABETIC FLOUR.

Total proteins	72.45 per cent.
Fat	0.7
Ash	6.30
Moisture	9.14
Carbohydrates	trace (less than 1%)

-----



During the period under review (Dec. 1919 to March 1923) there came under observation 54 cases of glycosuria with other symptoms. The following table shows the age, sex incidence, and mortality among these cases :-

Total cases.				
<u>Age</u>	<u>Male.</u>	<u>Known deaths before</u>	<u>Female.</u>	<u>Known deaths before</u>
		<u>1.iii.23.</u>		<u>1.iii.23.</u>
1 - 9	1	-	-	-
10-19	5	3	2	1
20-29	7	5	4	2
30-39	9	2	3	-
40-49	4	-	4	1
50-59	3	1	6	1
60-69	1	-	4	-
70-79	-	-	1	1
	<u>30</u>	<u>11</u>	<u>24</u>	<u>6</u>

30 male

24 female

54 cases of whom 17 (31%) were dead before 1.iii.23.

	<u>Male.</u>	<u>Female.</u>	
NOT diabetic	3	4	7
DIABETIC, but NOT suffering from diabetes.	1	1	2
DIABETIC and suffering from diabetes.	26	19	45.

In the cases classified above 7 are shown as being "not diabetic". One case was proved by glucose test to be a 'lag' - a girl of 21 (Case No.25) One man (Case No. 6) was probably of a similar type, but the glucose test had not been introduced at the time of his stay in hospital, and he could not, therefore, be properly classified. Certainly he was not a diabetic. The majority (males 2, females 3) were admitted to hospital for some

entirely different condition, and glycosuria was discovered on routine examination.

The second man (Case No. A.12) suffered from a purulent Otitis Media with glycosuria. The third man and three women (Cases A. 11 : 23 : 34 : 36) suffered from senility - 'done' old patients with arterio-sclerosis and glycosuria. The fourth woman (Case A. 39) suffered from obesity.

In the two 'lag' cases the glycosuria was negligible and required no special treatment. In the last case glycosuria disappeared on a moderately restricted diet, and did not recur on full diet after operation. In the senile patients the glycosuria was slight in amount, and was controlled or banished without trouble.

Of the two 'diabetic but not suffering from diabetes' cases, one was a woman suffering from pyelitis and cystitis who, on admission, was desperately ill with septicaemia. She died six days later. The other was a man suffering from Tabes Dorsalis with a strongly positive Wassermann Reaction. This last case and the 4 senile non-diabetic cases were treated on 'Allen' lines, and the response in every case was very satisfactory. All, save one who left irregularly, left hospital either free from glycosuria or with merely a trace of sugar in the urine. Doubtless in some, at any rate, of these cases some general instructions on restricting carbohydrate would have been sufficient to keep the urine sugar-free, but in most cases this had been tried without success by their own doctors, and, having got the patients

into hospital, it was obviously wise to give them some insight into proper treatment. On dismissal these cases were all instructed to report progress every three months.

The remaining 45 patients all suffered from diabetes of greater or less severity. In 3 cases death occurred shortly after admission to hospital. One case, already recorded under 'acidosis' died in coma within 48 hours of admission. Another case, a woman of 28, was admitted with air hunger and symptoms of acute cardiac failure, although quite clear mentally. She slowly sank into unconsciousness, and died 48 hours later. A third case, a man of 28, developed broncho-pneumonia a week after admission while under treatment, and died suddenly within 48 hours.

In the first two cases most energetic measures were adopted - oatmeal diet, abundance of fluid, intravenous sodium bicarbonate, stimulants - but all to no purpose. Indeed I have seen such treatment successful in one case only, the first I treated with 'Allen' diet. This man, a very severe case of diabetes with marked ketosis, developed slight air hunger after 3 months' treatment; but treated by the above method he made a remarkable recovery, and was able to leave hospital three weeks later (Case A1. ). The patient who developed pneumonia was put on to fluids and stimulant, but, when he appeared to be progressing favourably, he suddenly collapsed and died. (Case A.17)

Of the remaining 42 cases 12 were considered so acid

as to warrant commencing treatment with oatmeal diet. Two commenced on table I but subsequently developed an acidosis requiring oatmeal diet to clear it up. Five showed a moderate degree of acidosis and these, together with the remainder, 23 in number, went straight on to Table I, and ran their initial course according to its routine. In all, as I have explained above, routine treatment gave place later to adjustments to meet individual requirements. The obese were dismissed on diets insufficient for basal metabolism: the emaciated so far as possible on diets well exceeding this level: children on high calorie diets with a large protein content: manual workers on more liberal diets than sedentary workers. All were instructed to report frequently so that necessary alterations in diet might be made, and progress noted: but the immediate results of treatment were as follows :-

<u>Patients Treated.</u>	<u>Improved.</u>	<u>I.S.Q. or Worse.</u>	<u>Died.</u>
42	32	7	3

Reference has already been made to the three patients who died.

Of the 7 cases in the 'I.S.Q.' group ~~four~~ became dissatisfied with restricted diet and left hospital irregularly. One case had a progressive pulmonary tuberculosis on admission, and went to a Sanatorium: and one, a girl of 17, suffered from mitral disease with failing compensation. The sixth case, an elderly man with long-standing diabetes and gangrene of the toes, made no progress under treatment.

It is, however, apparent from the figures quoted above that the great majority of cases treated showed a definite improvement. But while the immediate prognosis was good experience showed that in hospital cases the end results were far from satisfactory. It was rare to find a patient of that class who did not lose ground after discharge from hospital. Almost all who were readmitted were found to have a distinctly decreased Carbohydrate tolerance in addition to a return of symptoms. Unwillingness to adhere to the diet was responsible in many cases, and inability to understand the rationale in a few. Only too often was one told that the diet was insufficient to allow work to be carried on: and, as loss of work was the alternative, diet was invariably increased in such cases with disastrous results.

Nevertheless patients could be kept going, even when the disease was severe in character, and where circumstances were far from favourable. The following two cases (Cases P.1. and P.2.) illustrate this point :-

		<u>1920.</u>	<u>1921.</u>	<u>1922.</u>	<u>1923.</u>
J.C.	Weight.	60 k.	61.6 k.	52.6 k.	59.7 k.
Age 42.	Carbohydrate Allowance	40 gms.	40gms.	60gms.	

This man carried on his work regularly as a plasterer save for these four admissions to hospital. The marked fall in weight in 1922 was due to a great exacerbation of the diabetic symptoms accompanying a septic hand: but he had almost reached

his old weight when admitted for insulin treatment in 1923.

	<u>1920.</u>	<u>1921.</u>	<u>1922.</u>	<u>1923.</u>
T.D. Weight.	29.5 k.	29.3k.	28.9k.	24.6k.
Age 13. Carbohydrate Allowance.	70gms.	30gms.	45 gms.	-

This boy did not adhere to the diets laid down for him, and, although a growing boy, his weight steadily fell; but he was kept alive until 1923 when he was admitted for insulin treatment. Indeed he was regularly at school until the end of 1922.

In March 1923 an attempt was made to discover the state of health of those patients who were alive.

Working and in good health	4 (m.3, f.1)
Readmitted for further treatment	14 (m.7, f.7)

Of these 14 patients 2 did not require treatment with insulin.

The other 12 patients showed signs of increasing difficulties, and were treated with insulin. Of these 12 patients 9 are alive and well, and 3 have died.

A girl, aged 15, died of phthisis.

A woman, aged 35, died of an acute broncho-pneumonia.

A woman, aged 64, died of gangrene of the feet.

No information as to the other patients could be procured.

Among private patients the results were, of course, much more satisfactory, although even here the restricted diet frequently proved in time too irksome. Frequent visits to report

progress were of vital necessity in all cases, and a word of encouragement was often wonderfully effective in reconciling a patient to his unhappy lot. It was not uncommon to find a zealous patient grossly underfeeding himself without regard to his general condition in an endeavour to keep the urine sugar and acetone-free at all costs. One patient, a man of 40 who had been under treatment in 1921 with good results carried on unaided until 1923, when persistent cough and spit brought him again under observation. He confessed to long periods of semi-starvation with deliberate loss of weight; but he was proud of the fact that he had remained free from glycosuria and acetonuria throughout. The general condition had greatly deteriorated, and there was an obvious tubercular lesion at the right apex (Case No. P.3.)

**INSULIN**

**TREATMENT**

**( )**



In the year 1889 Mehring and Minkowski demonstrated that complete extirpation of the pancreas in dogs was followed by severe diabetes which rapidly brought about the death of the animal. They also showed that the severity of the resulting condition varied with the amount of pancreas removed, and thus firmly established the "Pancreatic Theory" of the causation of diabetes. These results were confirmed by many workers. Very naturally the theory followed that relief of the condition in man was to be anticipated by the administration of fresh pancreas or extracts of the gland by mouth, and the striking success which had attended the treatment of myxoedema and sporadic cretinism with preparations of thyroid encouraged this belief. It was found, however, that expectations in this regard were not fulfilled, all preparations given by mouth being apparently destroyed in the stomach and rendered impotent. Capsules and keratin coatings were experimented with, but all without success.

Despite these failures work on gland replacement lines was not abandoned. As the result of numerous post mortem examinations evidence had accumulated that it was not the whole pancreas which was at fault, but the Islets of Langerhans - special groups of cells described by that observer in 1869. It was presumed that these cells furnished an internal secretion or hormone for which hypothetical substance Schafer suggested the name "Insulin." In 1904 Rennie and Fraser<sup>59</sup> prepared an extract from

the pancreas of certain fishes in which the islet tissue is distinct from the rest of the gland, and this extract they cautiously administered to certain cases. They noted some benefit in the majority of these patients, but the results were not sufficiently encouraging to cause them to persist, and they gave up, partly through lack of material. In 1906 Lydia de Witt attacked the problem. Taking advantage of the discovery by Schulze and Ssobolen that complete atrophy of the glandular structures of the pancreas followed ligation of the duct while the islet tissue remained unaffected, she prepared an extract of the islet tissue. This extract she found to possess glycolytic properties, but she could produce no benefit in diabetic patients by its administration. Her experiments were repeated in 1908 by Zuelzer<sup>12</sup>, using alcohol to extract the hormone. By this means he obtained a powder on evaporation of the alcohol, and this powder, dissolved in saline and administered intravenously, caused marked improvement in several diabetics by reducing both the sugar and the acetone bodies in the urine. The preparation of his extract was taken up commercially, but unfortunate toxic symptoms manifested themselves in many patients, while, generally, the results obtained were by no means satisfactory. The preparation was consequently dropped.

No further progress was made until 1922 when Banting<sup>5</sup> of Toronto took up the investigation. Using the technique of Lydia de Witt he repeated her experiments; but to prepare his extract he ground the partly degenerated pancreas in a mortar with sand and ice-cold Ringer solution. The extract prepared in this way

he tried upon depancreatized dogs; but, as I have explained in an earlier section, improved technique in the estimation of blood sugar placed Banting much more favourably for such a test, for now the effect of the extract could be observed upon the blood sugar, and not only upon the grosser changes in urinary sugar and acetone. The preparation, when injected into depancreatized dogs, was found to lower the blood sugar level and to reduce the sugar and acetone bodies in the urine. The problem had at last been solved, and Insulin had been isolated.<sup>42.</sup>

The next step was to discover some more simple method of obtaining the pancreatic extract. Having noted the discovery by Ibrahim that the pancreas of the ox contains no proteolytic enzyme up to the fourth month of intrauterine life, Banting, along with Best, prepared extracts from foetal pancreas, and again he was successful in producing a fall in blood sugar and a disappearance of acidosis. By either of these methods, however, only a very limited supply of insulin could be obtained. With the help of Collip there was devised a method of fractional precipitation with alcohol which gave from the pancreas of the adult ox a preparation of insulin free from any toxic properties. The process was rather complicated, but it depended upon the fact that insulin was not precipitated by 80% alcohol, but was precipitated by 93% alcohol, at which strength trypsin was destroyed. The insulin obtained in this way was standardised,

the standard being fixed as a "rabbit unit" i.e. a unit of insulin was that amount which on hypodermic injection lowered the blood sugar in a 2 kilo rabbit which had fasted for sixteen to twentyfour hours from the normal value of about 0.1% to 0.04%, thus causing typical convulsions within four hours. <sup>6, 28, 43.</sup>

The full details of this method of preparation were presented to the Medical Research Council in February, 1923, and by them the technique was made known to workers in various centres so that some experience of its preparation and use might be obtained before it was made generally available. In March, 1923, the first samples in Glasgow were prepared by Dr. Telfer in Professor Noel Paton's Laboratory, using Collip's technique. Large amounts of pancreas were used, but the yield of insulin was small. It was obtained in the form of a light brown powder, very hygroscopic, and readily soluble in 1/5 normal disodium-hydrogen phosphate. The solution of insulin thus prepared was standardised in rabbit units, and it was then handed to me for sterilisation before use. This I performed by passing the solution through a Massen filter, washing well with disodium-hydrogen phosphate solution. Thereafter the reaction of the solution was taken and small quantities of  $\frac{N}{10}$  NaOH were added to bring the reaction to about  $P_H$  7.5. A few drops of tricresol were then added, and the resultant solution was put up in vaccine bottles.

This method of preparation involved, of course, a very considerable dilution, so that I was rarely able to obtain a

greater concentration than one unit per c.c. of solution. This entailed a very large injection where big dosage was required, and compared very unfavourably with the commercial product of to-day whose strength is 20 units per c.c. But despite this disadvantage the insulin prepared in this way was used for four weeks, and in no case did any local reaction or general toxic effects follow its administration. It is greatly to the credit of Dr. Telfer that during much of this time our insulin was of greater strength and purity than that produced in most of the other centres of investigation.

By the end of April, 1923, insulin as a preparation called Iletin was obtained from America, and shortly afterwards a British product was placed upon the market. From that time forward I used only the commercial product.

E A R L Y   C A S E S.

The first case to come under treatment was a telegraph boy aged 16 years, who had previously been in hospital on three occasions (see Case P.2.) and with whom I had had little success, mainly through his misbehaviour in breaking diet. On each occasion he had shown marked glycosuria and acidosis; and, while each time he had improved under treatment, his condition had become steadily less satisfactory. He had been dismissed from hospital 11 months previously on an insufficient dietary and with persistent glycosuria (Chart III. Case P.1.) His doctor now reported that he had gone rapidly downhill of late, and that he was acutely ill. By a happy coincidence the first samples of insulin became available at this juncture, and the boy was re-admitted for treatment.

On admission he was terribly emaciated weighing only 24.5 kilo (3 st. 11 lbs.) The skin was dry and scaly, and so tightly drawn over the bones that it was with difficulty that it could be raised between the fingers. All the reflexes were absent, and the boy was incapable of moving himself in bed, while passive movement caused intense pain. The breath had a strong odour of acetone and <sup>the urine contained acetone and</sup> diacetic acid in amount. The urinary sugar output was 250 gms. in twentyfour hours, and the blood-sugar on admission was 0.5%. Insulin administration was commenced at once. Injections were very painful on account of the condition of the skin, and also because, as I have explained,

a comparatively large injection was necessary with the preparation which I had made. With a diet of C.170: P.60: F.70: he was given 10 units of insulin in the first 24 hours and 20 units in the following 24 hours, the diet on the second day being C.140: P.60: F.70. At the end of 48 hours the acetone had entirely disappeared from the urine, while the urinary sugar had dropped to 12 gms. in 24 hours. Meantime the skin had become perceptibly less dry, and the boy himself had begun to brighten up considerably. Insulin was continued in doses of 20 units, and gradually he began to move his limbs and head. Thirst and hunger became much less, and the urinary output dropped from 3,500 c.c. on admission to 1,600 c.c. His subsequent progress will be discussed in detail later.

The method adopted in controlling the giving of insulin to this patient is illustrated in Chart T.1. During the first six days (March 12th - 18th) the patient was allowed C. 130-170 (in reality quite as much as he was able to take) while insulin was administered night and morning, in insufficient dosage, as will be clear from the blood sugar estimations at this time, but in the only doses then available. On 19th March, however, there seemed to be sufficient insulin likely to come to hand to allow of larger dosage, and, as the patient was much improved, an attempt was made to discover the requisite dose on the diet allowed. To do this a dose of insulin was given followed thirty minutes later by a meal of known

C. P. and F. content (a "fixed meal"). Blood and urinary sugar estimations were performed much as in the "Glucose Test" described in an earlier section. Thus samples of urine and blood were obtained, a dose of 10 units of insulin was injected, and half an hour later (the optimum time then recommended by Banting) a meal consisting of C.20: P.18: F.25 was given. Thereafter hourly blood and urinary specimens were collected, and the results of these observations were charted as shown in Chart T.1.

The initial blood sugar was 0.21%: one hour after the meal it had risen to 0.24%, to fall in two hours to 0.17%, and in three hours to 0.09%. At four hours the blood sugar level was practically unchanged. The urinary sugar readings corresponded closely to the blood sugar estimations so that at three and four hours the urine was sugar-free. The dose in this case was not quite sufficient to keep the urine sugar-free, but, as it had reduced the blood sugar to a normal level, it was deemed wise to repeat the same dose before the evening meal. The meal on this occasion was C.28: P.22: F.30. Urinary sugar tests were negative hourly for four hours following this meal, while a blood-sugar test at three hours after insulin gave a reading of 0.11%. The balance of the C. P. and F. of the diet was spread over two other meals, one at 1 p.m. and one at 9 p.m. Next day these observations were repeated, but on this occasion the smaller "fixed meal" was given in the later part of the day, and, as the findings of the previous day had demonstrated that



the effect of the morning dose was apparently prolonged into the evening, the second dose was reduced to 8 units. On this day the urine remained free from sugar throughout, and the blood sugar, 0.12% in the morning, never rose above 0.18%. Three hours after the "fixed meal" in the evening it was normal.

By the following morning the blood sugar had risen to 0.38%. Evidently the evening dose of insulin had been insufficient to control the blood sugar level, and sugar was re-appearing in the urine. At this juncture the supply of insulin suddenly gave out, and, being left with twenty units only, this quantity was given on the third day in two doses of ten units, together with a similar diet to that of the first day. Four hours after the morning meal the blood sugar had reached a normal resting level. The intervening readings after the morning meal were much as in the earlier experiments, and urinary sugar estimations again gave a curve corresponding to the blood sugar curve. As before sugar was absent from the urine during the later part of the day, and the four hour blood sugar reading was normal. Next day (March 23rd) insulin was unobtainable, and blood sugar estimations made through the day showed a morning level of 0.21%, rising later to 0.30% on an unchanged diet. From this date until April 9th insulin was available only in insufficient amount so that I had to be content "to mark time", keeping the sugar excretion under control as far as possible.

Extracted from Author's Paper,  
Glasgow Medical Journal, August, 1923.

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Meetings of Societies.

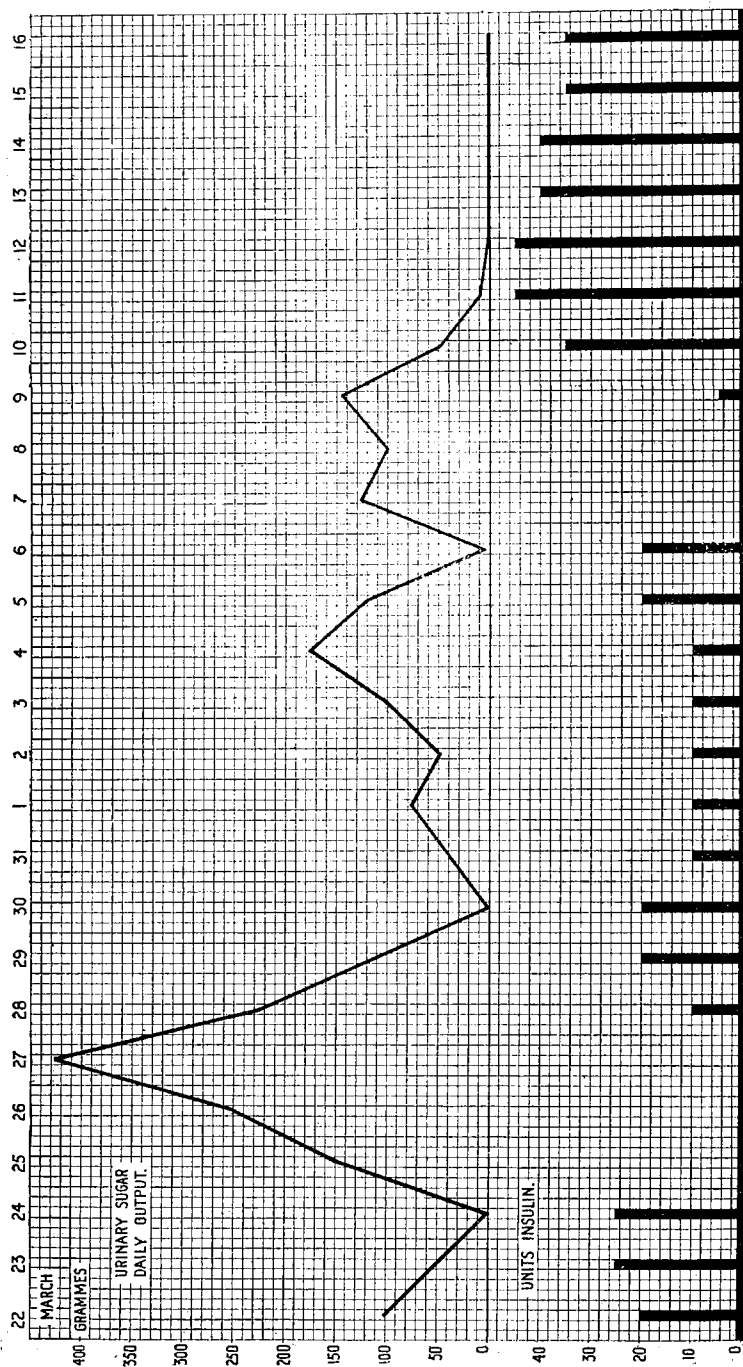


CHART V.

Chart D. shows clearly the effect of the withdrawal of insulin upon the urinary sugar output during this time.

On 9th April American insulin became available in adequate amounts and the investigations were recommenced. As I was uncertain concerning its strength as compared with my own (there had been some difficulty with the standardisation in America) I commenced with 5 units followed by a "fixed meal" as before. As previous experiments had demonstrated that the urinary sugar curve followed closely that of the blood sugar estimations, I somewhat modified the procedure. Specimens of urine were collected as before, but the blood-sugar observations were now reduced to two-hourly estimations, one at two hours and one at four hours after the meal, the intervening readings being deemed unnecessary. Later experience showed that a single blood-sugar four hours after insulin was a safe method of controlling dosage, while specimens of urine at two hours and four hours gave a sufficiently accurate check. Keeping the diet unchanged the dose of insulin was increased by 5 units to two doses of 10 units plus 10 units next day, and ~~on~~ on the eleventh to doses of 15 - 10. After 2 days on this dosage the urine became constantly sugar-free, and blood sugar readings were always within normal limits.

In this, the first case treated, a very large number of blood sugar estimations were made when insulin was first administered. Later, as I have explained, the number of these

estimations were reduced to, at most, a two hourly and a four hourly reading: but even this necessitated the restriction of treatment to a small number of cases. A further reduction was therefore necessary, but before proceeding with this modification it seemed wise to investigate the behaviour of the blood-sugar throughout the 24 hours, and for this purpose the following experiment was performed. The cases taken were :-

- (1) A normal healthy adult.
- (2) The case described above while on insulin in adequate dosage.
- (3) The same case with insulin omitted.

the diet in all three cases being the same over 24 hours. Twelve of these observations I made myself (10 p.m. to 9 a.m.), and I am indebted to Dr. Thomson and to Dr. Murray for the estimations performed throughout the day. So far as possible samples of urine were collected hourly, and the sugar content, if any, was estimated.

Charts X. Y and Z. show the results of this investigation. In the case of the normal adult the blood sugar ran within normal limits throughout the entire period. Slight rises occurred after each meal, but these were, on the whole, insignificant. The urine, of course, was free from sugar in every specimen.

(II) In the case of the diabetic under insulin the blood sugar readings again were all within normal limits, but the variations were much greater than in (1). The striking feature

was the presence of two marked depressions in the curve, each corresponding to the period following the administration of insulin, and each reaching its lowest point four or five hours after the injection. The efficacy of the second dose in checking the rising curve was also apparent. No substantial rise occurred in the curve until late in the evening, and then there was a rapid rise as the night advanced; but the blood sugar level never rose above the renal threshold, and thus the urine remained free from sugar throughout the 24 hours. This chart might therefore represent successful insulin treatment.

(III) The values in this chart were obtained by omitting insulin for a day in the same case while under treatment. Thus the initial blood sugar was only 0.15%. The first meal, however, raised the level of the curve, and, as we would expect from what has been said above, the return was not satisfactory. Thereafter the blood sugar rose steadily with succeeding meals till by the late evening a level of 0.34% had been reached. A fall then occurred, but throughout the night there was little change in the blood-sugar level, and no attempt was made to reach a normal reading. The blood sugar at 9 a.m. was 0.25% as compared with 0.15% on the previous morning.

Meantime urinary sugar analysis gave a curve which was very similar to that produced by the blood-sugar readings. Occasionally the patient was unable to pass urine and thus spaces occurred in this curve, but none the less the similarity

was very striking.

These three charts demonstrate the following points:-

(1) That in the normal individual on a low dietary the rise in blood sugar is very slight.

(2) That while no food is being given (8 p.m. to 4 a.m.) the blood sugar remains at a low level.

(3) That the blood-sugar in the diabetic is lowered by insulin and kept at a low level while food is being given by "protecting" (i.e. giving insulin before) the larger carbohydrate meals.

(4) That the blood sugar reaches its lowest level 4 - 5 hours after insulin.

(5) That insulin must be so arranged as to time of injection and size of dose that the rise of blood-sugar which occurs through the night will not be such as to bring that level above the renal threshold for the individual under treatment.

(6) That in the untreated diabetic the main rise in blood sugar occurs during the day when food is being given.

(7) That in the untreated diabetic the blood-sugar falls during the night, but in a case of any severity that fall is never such as to bring the blood-sugar to the normal resting level.

As a result of this investigation blood-sugar observations were reduced to a single estimation 4 - 5 hours

after insulin; but even this proved extremely inconvenient and very difficult to maintain regularly. The solution to the difficulty was found in the urinary sugar tests. These had been reduced to a test before insulin, and one two hours and four hours after insulin. A further reduction was made to two tests - one before insulin and one four hours after insulin. If sugar were present in the first specimen it could be assumed that the blood-sugar was high, and indicated that the usual dose of insulin would not be too great. If sugar were absent from the first test it indicated special observation lest the usual dose be too large: or it indicated a reduction in the dose. If sugar appeared in the second specimen then the dose of insulin was too small. If no sugar was present in the second then again the patient had to be carefully watched. To sum up - if sugar appeared in both specimens then blood-sugar estimations were unnecessary: but when both were sugar-free then a blood sugar estimation at four hours after insulin was essential for safety. Of course, this applied only during the earlier investigation period. When the dose of insulin was very cautiously advanced so that the urine was brought only gradually sugar-free, then blood sugar estimations were not required. Occasionally, as in Case In.2, a small carbohydrate meal was allowed about 10 p.m. in order to make the occurrence of a hypoglycaemic reaction very improbable, but generally such an addition was found to be unnecessary.

The first case which I treated out of hospital

was a Clerk, A.R., a man of 52 years, who had suffered from diabetes for two years. He had been on "Allen" treatment for 18 months when I first saw him, but he had been losing ground steadily. (This case is discussed in detail later). On 9/5/23 he was at work and apparently in fair health; but the urine contained 9% sugar, acetone, and diacetic acid. As he had been thoroughly investigated at a diabetic clinic elsewhere and the records were in my hands it was unnecessary to experiment further with "Allen" treatment alone, and insulin was commenced at once. I insisted upon a nurse being in attendance in order that he might be under skilled observation, especially during the night. As a preliminary measure I gave a meal of C.35: P.6: F.16., and hourly blood-sugar and urinary-sugar estimations were performed in the usual way. As chart T.2. shows the result was a typical diabetic curve, the blood-sugar varying between 0.28% and 0.38%. Meantime, as I had now come to expect, the urinary sugar curve followed a course similar to that of the blood-sugar curve. The following day I repeated the same meal with 10 units of insulin half an hour previously. The blood-sugar rose from 0.28% to 0.31%, to fall in four hours to 0.168%. A similar fall was found in the urinary sugar. In the evening I repeated the meal (C.35: P.6: F.16) the remainder of the total dietary of C.75: P.90: F.90 had been consumed at mid-day) preceding it on this occasion by 15 units of insulin. The blood-sugar, 0.20% before the meal, fell to 0.08% in four hours, while the urine became sugar-free. Shortly after the last blood-sugar



had been taken the patient complained of some general nervousness with palpitation and marked sweating. A cup of hot unsweetened tea and 10 gms. of white bread were given immediately, and the discomfort passed off in about 5 minutes. As the patient was a distinctly nervous man and I had warned him what to look for in case of an overdose of insulin, I was uncertain whether this was a true hypoglycaemia or not; but in view of the low final reading I thought it wise to give a small amount of carbohydrate to prevent alarming symptoms. No further developments occurred. Next day, however, I reduced the insulin to 10 units for the same meal. The initial blood-sugar was 0.175%: four hours later it was 0.225%. Meantime the urine, which had been free from sugar in the morning, showed 8% of sugar. To be safe I gave only 5 units in the evening with, as is clear from the chart, very unsatisfactory results. The following day I reverted to 10 units of insulin with a fairly good result; but this dose, repeated in the evening, was again decidedly unsatisfactory. On this day and the following day I performed only one blood-sugar estimation, that four hours after the second dose. The question of expense had to be considered, and, moreover, as in the infirmity cases reported above, the urinary-sugar percentage curves followed the blood-sugar curves so closely as to render the latter unnecessary. Only when the urine became constantly sugar-free did a blood-sugar test seem to be requisite, and on this reasoning I proceeded. Experience subsequently showed

this to be correct. To prevent the occurrence of a hypoglycaemic reaction through the night, or at any rate to render such a happening much less probable, I increased the diet to C.90: P.90: F.100 to allow of a small meal being taken late in the evening just before going to bed. To continue, I now increased the insulin at each dose by 5 units, giving the larger dose before the morning meal, with the object of bringing the urine sugar-free throughout the 24 hours. Thus on succeeding days I gave units 20 + 15: 30 + 20: 30 + 20: 30 + 25: and 35 + 30. At this last dosage I halted. The sugar in the urine showed a steady decline in amount, and after four days on 65 units it disappeared completely. A blood sugar four hours after the second dose gave a reading within normal resting limits.

Chart T.3. shows the treatment of another private case at a later stage in the investigation. This patient had been on "Allen" treatment for several years, but his tolerance for carbohydrate had steadily declined, and when treatment commenced he was far from well. The urine contained 280 gms. of sugar and acetone +. In this case I commenced on a fixed diet C.90: P.80: F.90 giving, as before, the bulk of the carbohydrate in a morning and evening meal preceded by insulin. The dose of insulin was increased rapidly from units 10 + 15 to units 35 + 30, but later it was reduced to 30 + 25 and continued at this level. Few blood sugar estimations were made and urinary samples were collected only before insulin and three hours after the meal. As in the earlier cases a gradual fall in the urinary-sugar content occurred until finally the urine became constantly sugar-free.

These three charts show very clearly, I think, the method which I adopted in the treatment of the earlier cases. I had always in view two objects -

(1) to treat the patients safely, i.e.

with a minimum of risk of a

hypoglycaemic reaction

- and -

(2) to simplify the treatment as far as

possible so that a scheme of treatment might

be evolved which did not require elaborate

methods of investigation and might thus be

readily carried out in private practice

without recourse to hospital treatment.

It was with this second end in view that I strove to reduce to a minimum the number of blood-sugar estimations which seemed necessary.<sup>27,35</sup> From the charts above it is clear, as I have already stated, that so long as sugar is present in the urine blood-sugar estimations are unnecessary; but when the urine becomes sugar-free such a test four or five hours after the second dose of insulin should be performed whenever possible.<sup>64.</sup>

"P E D I G R E E" D I A B E T I C S.

So far I have dealt exclusively with my experience with the earlier cases and with the methods adopted in bringing these patients on to full treatment, i.e. on to an adequate diet with such a dosage of insulin as to prevent glycosuria. The patients referred to were all diabetics of standing - patients who had been under treatment for several years and who were known to require insulin as soon as that drug became available. That there was a considerable number of cases which had previously been under treatment and were alive at the time of the introduction of insulin is clear on referring to Table I in the "Allen" Section. Of the 55 "Allen" cases 17 were known to be dead by March, 1923. So far as could be discovered the cause of death in these cases was as follows :-

	<u>Male.</u>	<u>Female.</u>
Diabetic Coma (Following irregular dismissal in 3)	4	-
Phthisis	1	1
Pneumonia	1	-
Appendicitis	1	-
Pyelitis	-	1
'Cardiac Failure'	-	2
Probably Diabetic Coma	1	1
Unknown	3	-
	<u>11</u>	<u>5</u>

The remaining 38 were written to and asked to report for examination, but only 18 responded to this invitation. Of these 18 patients 4 were found to be in excellent health, satisfied with their diets, maintaining their weight, and well able for

their work. The remaining 14 were found to have lost ground to a greater or less degree and all were re-admitted for treatment. These formed three groups :-

- (a) Those who had deteriorated little, but who had lost weight, and whose urine showed sugar and acetone in slight degree.
- (b) Those who had lost much weight and were emaciated, and whose urine generally showed sugar in amount and acetone in varying degree.
- (c) Those whose nutrition was fair, but who showed evidence of marked acidosis - acetone in the breath, and acetone and diacetic acid in the urine.

Group (a) consisted of 6 cases. One woman (Case P.10) was admitted with haematemesis, and her treatment was the usual one for this condition. During her residence, however, sugar never appeared in the urine even on a liberal diet, and thus she ceased to be regarded as a diabetic. No satisfactory explanation of this curious phenomenon has yet been advanced. The remaining 5 patients commenced treatment on the older starvation method with a rebuilding of the diet on the usual "Allen" lines, and every effort was made to treat without insulin. At that time insulin was very expensive and no provision had been made for its supply out of hospital. Again, its administration by injection to be given either by the patient himself or by some relative presented a very real problem in patients of the infirmary class, while the dangers of overdosage in these circumstances had also to be remembered.

Thus every effort was made to treat on diet alone, but

in no case was this successful. One patient (Case P.11) an old lady with gangrene of the toes, received only a few doses of insulin, but with little benefit. A girl (Case P.14) who appeared to be progressing satisfactorily developed a 'lighting up' of an old apical tubercular lesion with a return of diabetic symptoms, and insulin was required to maintain nutrition. The remaining 3 cases all showed a marked decrease in carbohydrate tolerance, and it was found impossible to raise the diet to an adequate calorie value without insulin. One case (Case P.9) was dieted at home and came into hospital for insulin. The progress of one of those cases is typical of many which follow, and is illustrated in chart R.

It will be seen that the urine on admission contained a considerable amount of sugar, and acetone +. On a decreasing diet the urine was readily rendered sugar-free; but increasing diet showed the tolerance to be low, and the diet, after 3 weeks' treatment, was inadequate for his requirements. Any attempt to increase the diet was followed by glycosuria. Meantime the patient had lost almost half a stone in weight. Keeping the diet at the level which had caused glycosuria to occur (in this case C.80: P.70: F.100) I commenced insulin in 5 unit doses. The dose was increased fairly rapidly in this case - ten units, fifteen units, twenty units on succeeding days, and on the last dose the urine became sugar free. Thereafter the diet was in turn increased, again in this case

rather rapidly as time was a consideration and the man had to return to work or lose his position. But sugar did not reappear in the urine and the patient left hospital on an adequate diet, showing an increase in weight, and on 20 units of insulin.

I take this case as an example of the method generally adopted in the treatment of diabetic patients of moderate severity, save that in regard to insulin dosage I advanced generally more slowly, as will be clear from the charts shown below. To other methods I shall refer later.

Group (b) Patients showing much emaciation. 5 of the 14 cases re-admitted for treatment were so emaciated as to render immediate feeding necessary. Frequently, too, they showed signs of cardiac failure, and this was met by stimulation and by the usual cardiac tonics - alcohol, digitalis, caffeine, &c. The procedure adopted was to place each case on admission upon a calculated adequate diet arrived at as described in a previous section. Insulin was commenced at once, the dose being cautiously increased as previously described. This process was continued until the patient became free from glycosuria, when diet and insulin remained unchanged for as long as was considered necessary. Thus a patient of 4 ft. 2 in. in height but weighing only 22.5 k. was put upon a diet of C.60: P.40: F.50 the calorie value of which is 710 C. 3 days later the diet was increased to C.60: P.45: F.100 where it remained constant. This represents the calorie requirements of a lad



not of his present weight, but of a lightly built boy of the same height. The bulk of the carbohydrate was divided between the morning and evening meals, and insulin commenced in a dose of 5 units half an hour before one of these meals. The dose was then increased by 5 units before each meal until the urine became sugar-free, when no further increase was made. Chart S. illustrates the course of a case treated on these lines.

Group (c) Patients showing severe acidosis.

This group consisted of 3 cases. The first essential in these cases was to eliminate the acidosis, and to this end insulin was given in large doses together with a diet containing a certain amount of carbohydrate. The largest dosage of insulin required in these cases (Case P.12: last admission) was 60 units in 24 hours, but in the other 2 cases 10 and 15 units respectively in 24 hours was sufficient. Again the insulin was rapidly advanced. All 3 cases were placed upon diets rich in carbohydrate, and with a low fat content. Subsequently, in less 'acid' cases, various diets were experimented with but a modification of the old van Noorden oatmeal diet appeared to be the most efficacious for the purpose. This diet was really that of van Noorden without eggs or butter, and has been described in the "Allen" Section under "Acidosis". Opinion is divided as regards the wisdom of giving sugar in these cases, and, as Maclean<sup>38</sup> has pointed out, the blood sugar is invariably so high that further carbohydrate seems contra-indicated. But

Joslin<sup>31</sup> and others<sup>52</sup> consider that the carbohydrate assists the action of insulin in the metabolism of fat, and my experience confirms this view. Abundance of fluid is essential, and, if the gruel could not be tolerated as occasionally happened, water, tea, coffee and milk were given in its stead. If nothing could be taken by mouth enemata or subcutaneous and intravenous salines would have been employed; but this was never necessary in any of the cases.

It will readily be understood that when employing such large doses of insulin frequent blood-sugar observations were necessary, although, as with smaller doses, frequent testing of samples of urine formed a very reliable guide to progress. But the fall in blood-sugar level may be very rapid, and, as the life of the patient may depend upon adequate dosage, caution can hardly be advocated. Thus although in these cases particularly are blood tests essential, none the less insulin should never be withheld merely because they cannot be performed.

Chart U. illustrates the treatment of an "acid" case. On admission the blood sugar was 0.255% and the urine contained much sugar (220 gms. in the first 24 hours) and acetone ++++. The patient received 10 units of insulin on the evening of admission with 60 gms. of carbohydrate. During the following 24 hours he had 30 units with a diet rich in carbohydrate, the acetone being somewhat less marked in the urine and oatmeal consequently considered unnecessary. On increasing insulin to a maximum of 50 units and decreasing carbohydrate the urine was

brought acetone-free in 10 days. Thereafter adjustment of insulin to dietary requirements followed the usual methods.

It is convenient to state here briefly what I regarded as an "acid" case requiring immediate insulin treatment, and how I differentiated this from one less urgent which might safely be treated by starvation. In the "Allen" section I have detailed the various points to note, odour of acetone in the breath, presence of acetone and diacetic acid in the urine in amount, low alveolar  $\text{CO}_2$  and raised ammonia-nitrogen ratio. To this I added the Van Slyke<sup>65</sup> method of estimating the blood bicarbonate. This method is extremely ingenious and is performed without difficulty. A sufficient degree of skill can readily be attained for reasonably accurate observations. On the whole I did not find it very helpful, although, when taken in conjunction with the other observations it strengthened the picture. Speaking generally, as in the days of "Allen" treatment, I found it much wiser to treat any doubtful patient as serious and to commence insulin administration at once. Of course, with insulin in reserve there is not the same risk attached to injudicious treatment as during the "Allen" period, but the insulin could be used as a temporary expedient to clear the acidosis and could be discontinued later if circumstances allowed. There was thus no contra-indication to its use, and further experience has gone to show that this is sound practice. (see "New Cases".)

## RESULTS OF TREATMENT.

While the results of treatment in the "Pedigree" diabetics will be considered later together with the results in the "New" Cases, there are one or two points which they alone illustrate.

All the patients in this group (save the anomalous case P.10) were cases of severe diabetes, and many had shown but a poor response to "Allen" treatment. 7 of these had suffered from more or less severe relapses since their first admission to hospital, and they had been readmitted on one or two occasions for further treatment. All had shown glycosuria in amount when readmitted, and several had shown evidence of marked acidosis. Speaking generally the trend had been downhill. Case P.2. is typical of the group, and will serve as an example.

### Hospital admissions:

	<u>1920.</u>	<u>1921.</u>	<u>1922.</u>	<u>1923.</u>	<u>May.</u> <u>1925.</u>
Weight (Chart W)	29.5k.	29.5k.	24.4k.	24.4k.	43k.
Urinary Sugar	40gms.	138gms.	64gms.	252gms.	Free.
Urinary Acetone	++	++++	+++	++++	-

This table illustrates the condition of the patient on admission on 4 occasions. On the fourth occasion he was admitted for insulin treatment. Although he was a growing boy the weight had fallen 5 kilos in 3 years: indeed he was literally skin and bone. In two years, with insulin, he has practically doubled his weight. After dismissal in 1923 the patient did not require to be readmitted, and the observations in May 1925 were made

when he came up to report. Indeed, from the introduction of insulin in March 1923 until December 1924, no patient required to be readmitted for treatment save two, one (Case P.6.) who developed a carbuncle, and one (Case P.12) who died of Broncho-pneumonia. Thus under insulin relapses have been infrequent, and hospital treatment was seldom necessary even in the most severe cases. The contrast with the "Allen" period is worthy of note.<sup>53</sup>

The effect on Carbohydrate Tolerance was striking in 13 "Pedigree" cases (omitting case P.10)

	<u>Gain.</u>	<u>Loss.</u>	<u>Unchanged.</u>
Male	5	2	1
Female	4	1	-

The gain in carbohydrate tolerance was fairly constant. One case gained 105 gms. The average gain was 41 gms. The unchanged case was one complicated by a carbuncle. The three 'loss' cases showed respectively a drop of 40, 20 and 70 gms. All 3 had carried on without proper supervision for about 2 years, and their tolerance was so reduced in consequence that even with insulin it could not be raised to its earlier level. (Cases P.3: P.4: P.9.)

It might be expected that in these "Pedigree Cases" the beneficial results of insulin treatment would be most striking. So far as immediate results are concerned this is true only in the very 'acid' or very emaciated cases, and particularly, as in case P.2., where the patient was 'in extremis'. In such cases

the results are almost dramatic in their sudden appearance.<sup>4, 57.</sup>  
In almost every case, too, acetone and sugar are rapidly cleared from the urine, with consequent disappearance of many symptoms and much increased comfort to the patient. But such changes as increasing weight and returning vigour do not manifest themselves until later. To these points I shall refer in considering the results of insulin treatment in all cases in a later section.

SELECTION OF CASES FOR TREATMENT.

With regard to patients admitted for the first time with complaint of glycosuria, the point of primary importance was to determine the nature of this symptom. The method adopted for this purpose has been fully explained in Section II of this paper, and requires no elaboration here. It will, however, be quite clear from what has been said concerning the power which insulin possesses of reducing the level of sugar in the blood that "renal" and "lag" cases were not only quite unsuitable for insulin treatment, but would be exposed to considerable risks by such treatment.<sup>2,68</sup> Fortunately these patients rarely complained of typical diabetic symptoms, save, perhaps some loss of weight, and thus there is little likelihood of their being regarded as diabetic, even when blood sugar estimations cannot be made. In the cases under discussion here blood-sugar observations were invariably taken, and the true nature of each case was determined: but a simple test carried out on case No. ~~46~~ **Ix.46** and illustrated in chart **W** demonstrates how, without blood-sugar tests, a diagnosis of a non-diabetic glycosuria may be indicated. In this case the carbohydrate intake varied between 45gms. and 160gms. in the 24 hours. The urinary sugar output varied very little, and did not change in perfect correspondence with the ingestion of carbohydrate as almost always occurs in the diabetic. It is not suggested that this method is in any way conclusive, but, where such a condition is

found, insulin should never be administered..



NEW CASES.

During the period March 1923 to Dec. 1924 there were admitted for treatment 81 cases. Table II shows the age and sex incidence, and mortality among these cases.

Table II.

<u>Age.</u>	<u>Male.</u>	<u>Deaths.</u>	<u>Female.</u>	<u>Deaths.</u>
1 - 9	2	-	1	-
10 - 19	5	1	2	1
20 - 29	4	-	5	-
30 - 39	9	-	5	1
40 - 49	9	-	6	1
50 - 59	9	-	11	-
60 - 69	7	2	5	1
70 - 79	-	-	1	-
	<hr/> 45	<hr/> 3	<hr/> 36	<hr/> 4

81 cases, of whom 7 were dead before Jan. 1st, 1925.

"Pedigree Cases" = 14

"New Cases" = 67	{	Insulin at once	-	18
		" later	-	22
		No insulin	-	27

The treatment of cases admitted for the first time varied a little, but certain general principles were followed.

In every case, save where acidosis or emaciation caused the immediate use of insulin, treatment commenced on the older dietetic lines described in the "Allen" Section. Every effort was made to avoid the use of insulin. In his more recent writings Joslin<sup>34</sup> states that he now very rarely starves a patient. Personally I think this is a mistake. The disciplinary training which a patient receives at such a time is invaluable, and experience has shown that it is in patients who have been under such training that the best results with insulin are achieved should its use later become necessary. Again insulin administration is troublesome. It demands unremitting care and attention to detail. A fair standard of intelligence is required for its safe administration over long periods. Food-stuffs have to be as carefully weighed as under the older "Allen" method: and very frequently the additional food allowance is no fair compensation for the trouble involved. Moreover, patients on insulin may go very far wrong before the necessity for a careful scrutiny of their mode of life becomes apparent. The power which insulin possesses of keeping acidosis in check while glycosuria may become considerable and weight is lost deprives such patients of many timely warnings - sweet odour of the breath, sweet taste in the mouth, drowsiness, &c. It may be argued that urinary sugar tests and records of weights will prevent this, but the vast majority of patients grow careless as time goes on, and very generally these precautions are over-

looked. Thus where possible insulin is to be avoided. Where the patient could reach an adequate or equilibrium diet for the work which he has to perform it was invariably withheld, and very rarely did any urge its use when its disadvantages had been properly explained. Many, of course, experienced acute disappointment. The newspaper campaign had led them to believe that a simple and speedy cure had been discovered, and their chagrin upon learning the true facts of the case was very great, and had a most depressing effect upon them.

The total number of "new" patients admitted for treatment was 67. The significance of the glycosuria in these cases varied as follows :-

	M.	F.
"Renal" Glycosuria	1	2
"Lag"	2	-
Diabetic, without symptoms of diabetes.	4	4
Diabetic, with symptoms	30	24

The 3 Renal and 2 Lag cases (Cases In.29: 41: 47: 15: 33) were all confirmed by one or more Glucose Tests, and were dismissed from hospital with very little restriction upon their diets. Of the 8 diabetic patients without symptoms the 4 men suffered respectively from Acute Phthisis, Chronic Interstitial Nephritis, Syphilis, and Tabes Dorsalis, ( Cases In. 26: 25: 31: 4). The 4 women were cases of Cystitis, Cystitis with Rheumatoid Arthritis, Rheumatoid Arthritis, Senility, (Cases In. 41: 49: 56: 40). All were confirmed by Glucose test or by blood sugar estimations, and all were treated as under the "Allen" regime, by a moderate restriction in diet, due consideration being given

to its suitability  
in the various ailments.

54 Patients suffered from diabetes with symptoms. 36 of these commenced treatment on the usual dietetic lines. The remainder, 18 in number, required insulin on admission.

Of the cases which received insulin on admission 13 were so treated on account of acidosis. In 4 of these an oatmeal dietary was also considered necessary; in 10 a diet rich in carbohydrate was given. 4 cases were so emaciated that treatment commenced with insulin and a full diet. The treatment in these 2 groups was identical with that described for similar cases in the "Pedigree" series. The eighteenth patient was an old man with gangrene of the toes who was neither emaciated nor showing evidence of acidosis. The insulin was given in this case in the hope of checking the spread of the gangrene and expediting the healing of the toes.

14 cases received no insulin treatment. One of these patients left hospital irregularly before the severity of his condition could be determined. The remaining 13 progressed very satisfactorily on diet alone, and thus insulin was unnecessary.

In the patients who were found to require insulin to allow of diets adequate to their requirements, the majority were treated as described under the "Insulin Later" group in the "Pedigree" series: i.e. the diet was advanced as far as possible without insulin, and then insulin administration followed the gradually increasing diet until the requisite diet and dose were reached. A few patients were treated in one of two recognised methods

(2 by method (1): 3 by method (2)).

The first method was that advocated especially by Graham<sup>20, 21, 62.</sup> in which a minimum of extra carbohydrate and of insulin were given, every advance being cautiously made and particular attention being paid to a normal pre-breakfast blood-sugar reading. Undoubtedly this was a most satisfactory method of treatment and an endeavour was made to treat the early cases and the younger patients on these lines. Fortunately the blood and urinary sugars are more readily controlled in such cases and thus they were particularly suited to it. This method, however, demanded a rather long residence in hospital for which most patients could not afford the time.

The second method was one which Maclean<sup>39</sup> recommended, and was similar to that described under "Emaciated" Cases in the "Pedigree" series. In this a calculated adequate diet for the patient was selected, and he commenced with this diet as soon as it was decided that insulin was necessary. Insulin was now administered in gradually increasing doses until the urine was brought sugar-free and the blood-sugar constantly within normal limits. For the long-standing case and for the elderly this was the method of choice; but for the average hospital case it was considered to be more satisfactory to adopt the method which I have described above. The cases which follow give numerous illustrations of the success of this method.

Insulin was occasionally employed to expedite treatment in certain cases. A stout young woman (Case No. In.45) was

admitted with much sugar and acetone in the urine. She would probably have progressed quite satisfactorily on diet alone, but much difficulty was experienced in eliminating sugar and acetone from the urine, and it was imperative that her stay in hospital be short owing to home circumstances. Small doses of insulin were given and continued for a week. Acetonuria and glycosuria quickly disappeared, when insulin was stopped and subsequent treatment proceeded on the usual dietetic lines.

Recently this procedure has been employed more often, but it should be used only where, as in hospital, the patient is under careful observation, and where progress can be checked by blood sugar estimations.

Such then were the lines upon which diabetic patients were treated between March 1923, and December 1924. Some further points now fall to be considered in greater detail.

D O S E   O F   I N S U L I N .

The dose of insulin varied very greatly. Apart from the cases which died the highest dosage given in any case was 85 units in the 24 hours. The average maximum dose was 33.5 units: while the average dismissal dose was 21.2 units, the amounts varying from 55 units in 2 cases to 0 units in 8 cases. In all cases where insulin had to be carried on after discharge diet was so arranged as to allow of, at most, a morning and evening injection. The objections to a mid-day dose in the case of men at work in offices and at trades are obvious. It has even been found more expedient, in elderly patients at any rate, to permit a slight glycosuria rather than to insist upon a more frequent dosage. Some patients tend to lose heart and to abandon treatment in despair when too many injections are demanded of them. Many of the older patients were made comfortable and able to carry on merely by keeping their sugar excretion in check with insulin. Several examples of this procedure are given among the "Insulin Cases" below, e.g. Cases In. 5; 6. In younger people and in early cases, however, such a state of matters should never be permitted. It is of primary importance that such patients remain sugar-free throughout the entire day.

Recently I have introduced a system of increased dosage in certain cases. Not infrequently patients are found to

show sugar in the urine in the afternoon although free from glycosuria throughout the remainder of the 24 hours. A third dose at mid-day keeps the urine constantly sugar-free. In these cases I advocate the use of three doses on Saturday and Sunday, when the third dose can conveniently be given at home. The results have been very satisfactory since not only is the patient free from glycosuria on these days, but the effect is prolonged into the following week, and generally there is no return of glycosuria until late in the week - say Thursday or Friday.

Mention might be made here of another method which I have tried, and which was suggested to me by Murray Lyon of Edinburgh. A patient on a fixed diet and fixed insulin dosage is starved every Sunday, but the insulin dosage remains unaltered upon that day. The result is, of course, to lower the blood-sugar level very markedly, and so to place the patient very favourably for the following week's diet. Hypoglycaemic reactions, however, are apt to occur upon the starve day, and, having experienced two such happenings, I gave up this method.

The dose of insulin in every case should be the minimum dose required to keep the patient's urine sugar-free on an equilibrium diet. If it is considered wise to increase the patient's weight then the dose should be increased to permit of a more generous diet; but when the increase in weight has been brought about then diet and insulin dosage



should be at the minimum to maintain this weight. As under the "Allen" regime overfeeding should be strongly discouraged, and under no circumstances, save perhaps occasionally in elderly patients, should increased insulin be given to make it possible. As Joslin<sup>31</sup> quaintly expresses it, "the patient (on large doses) is walking on longer insulin stilts, and his equilibrium is therefore correspondingly endangered". Again, with small doses there is less induration at the sites of injection, an important matter where insulin has become, as far as one can see, a permanent routine. With small doses, too, there is always the feeling that something is held in reserve should time prove that larger doses will become necessary. So far anxiety on this score has proved without foundation, no case now alive in the 47 (54 treated: 7 dead) quoted here requiring to-day a larger dose than on dismissal from hospital so far as I have been able to discover. Indeed, several patients have been compelled to reduce the dose, especially when engaged on harder manual work, or when taking much exercise. Nevertheless the knowledge that a considerable increase could be made is very comforting to both patient and physician.

Geyelin has stated that there is "no evidence that the degree of carbohydrate tolerance which any case of diabetes can achieve with insulin is limited by anything except the amount of insulin which can be given".<sup>31</sup> Personally I feel satisfied, although it is rather difficult to prove, that there is an optimum dose in the case of each patient. In case In. (1),

for example, the dose of insulin at one period varied from 55 to 85 units on a constant diet with little change in blood-sugar readings or in sugar excretion. In a few other cases in which big doses have been required a similar result has been noted. These were, however, all earlier cases where the standardisation of the product was not so reliable as to-day, and this factor may explain these differences. Latterly I have not given such large doses as those recorded above, preferring rather to persist with a smaller dose until glycosuria was abolished, thus I have not had the opportunity of repeating these observations. None the less they seem to me to suggest at least that there is an optimum dose in each case, and that insulin, beyond a certain limit in each case, loses in great measure its efficacy.

TIME OF INJECTION.

Originally the injection of insulin was given half an hour before the meal it was intended to "protect". This was the optimum time recommended by Banting in 1923, and for many cases it is the most suitable interval. Some, however, appear to do better when the interval is prolonged.<sup>36</sup> In one case it was found by experiment that 90 minutes was the most successful interval: in two others it was one hour. It is my practice to commence with a half hourly interval and subsequently to experiment with several variations in case a more suitable time may be found. The average interval between insulin and meal in the cases here recorded was 34 minutes.

RESULTS OF INSULIN

TREATMENT.

RESULTS OF INSULIN  
TREATMENT.

7 Cases died during the period March, 1923, to Decr. 1924, 2 male and 5 female.

4 Patients died at home, 1 male and 3 female. The man and one woman suffered from arterio-sclerosis and gangrene of the toes in hospital, and had been little benefitted by their residence. They died shortly after returning home. The cause of death in the second <sup>and third</sup> woman is uncertain, but was probably coma.

Of the patients who died in hospital the man was known to have had diabetes for 2 years, and on a previous occasion coma had seemed imminent, but had been averted. He had been in bed for 2 weeks before admission and had become very ill the previous day. On admission he was collapsed and cold. The pulse was rapid and poor in quality. The respirations were frequent and sighing in character. The breath smelt strongly of acetone, and he was very drowsy. The blood-sugar was 0.265%; the urine contained 5% sugar, acetone ++, and a heavy cloud of albumen. There was a marked lipaemia. Under stimulation and with insulin, 45 units, the pulse improved, he became more intelligent, and the drowsiness practically disappeared; but the pulse gradually failed and he died 12 hours after admission from cardiac failure. (Case In. 14.)

1 woman who had previously been treated in hospital and who was carrying on with insulin outside, was admitted with broncho-pneumonia. The urine contained 2.7% sugar, and acetone +. The blood-sugar was 0.40%. There was no suggestion of drowsiness, and the respirations were rapid and shallow. Under insulin, 90 units, the acetonuria practically disappeared in 48 hours, but she died of cardiac failure, mentally clear to the end. (Case P.12)

The second female was a girl who suffered from diabetes of a moderately severe type, together with an extensive tuberculous lesion of the right lung. Treatment of the diabetes with insulin was very satisfactory, but she had several severe attacks of Haemoptysis while in hospital. <sup>58</sup> On dismissal she was transferred to a sanatorium, where she died shortly afterwards. (Case P.14).

In reviewing the general results of treatment the "Pedigree" and "New" groups may be considered together; but it must be remembered that the severity of the condition varied in the "New" group of cases, although the majority were of a severe type. Of the total cases 54 treated with insulin 3 died in hospital; the remaining 51 without exception showed some improvement under insulin. Thirst and polyuria disappeared rapidly in all cases. Pruritis was rarely troublesome. The change in the mental outlook was often very striking, and an appearance of general well-being replaced the dejected air of the chronic and hopeless

invalid. Frequently even while in hospital the eyesight improved to some extent. One case of toxic amblyopia benefitted very greatly by insulin treatment. (Case In. 7) This patient was sent to me by an Occulist suffering from toxic amblyopia, due either to tobacco or to diabetes. His sight was so impaired that he was unable to read the largest of print even with lenses. After 6 weeks on insulin he was able to read the newspaper with suitable glasses. As he confessed to having continued to smoke 7 ounces of tobacco per week throughout the improvement would seem undoubtedly to have been due to insulin.

In 2 cases (Cases P.3. and In. 2) the knee jerks were absent on admission, but returned after a period of insulin treatment. A gradually increasing excitability in this reflex as treatment proceeded was a fairly common finding.

The effect of treatment upon weight was rather surprising:-

#### WEIGHT.

The weights in both groups varied as follows :-

	<u>Gain.</u>	<u>Loss.</u>	<u>Unchanged.</u>
Male.	13	11	10
Female	7	6	7

From these figures it is clear that in the majority of cases the weight either remained unchanged or fell while under treatment in hospital. The significance of this point

is recognised when it is remembered that the great majority commenced treatment on starvation diets. The greatest loss of weight was 4 kilos, and the average loss 1.9 kilos. The greatest gain was 10 kilos, occurring in an emaciated patient admitted with a carbuncle on the shoulder. (Case P.6.) The average gain was 2.7 kilos. A gain in weight was more frequent among the younger patients. In 6 patients below the age of 20 a gain in weight was recorded in 5. The sixth suffered from pulmonary tuberculosis.

A "follow-up" of these cases, however, has revealed a much more satisfactory result. In all who have carried out treatment conscientiously a gain in weight has been invariable. 2 women, one a clerkess, the other a mill-worker, (Cases In. 40 and In. 49) gained 5.5 kilos and 8 kilos respectively in the 3 months following dismissal. A boy, (Case P.2.) who gained 5 kilos while in hospital, gained an additional 9 kilos in 6 months. He had been in regular employment as a clerk. Case In. 2 has gained 6.5 kilos ; Case In. 13. 4.5 kilos; and many examples of similar results could be quoted. Unfortunately all trace has been lost of some of the infirmary cases, but in 27 patients who have reported recently there has been an average gain in weight of 2.2 kilos.

The effect of insulin upon carbohydrate tolerance has already been discussed. Only very occasionally has an attempt been made to increase the carbohydrate tolerance



after dismissal. Where improvement has occurred insulin has been reduced either in amount or in the number of injections required. In 2 cases where such an improvement in tolerance has occurred the patients have regarded the reduction of insulin as the greater boon. So far as my experience goes an appreciable gain in carbohydrate tolerance after prolonged insulin treatment is rare, even among the early cases or younger patients. Certainly the carbohydrate tolerance has never been raised to a level which could subsequently be maintained without insulin. Case P.6. is instructive in this respect.

In July 1923 I reported upon this case.<sup>60</sup> Under insulin his carbohydrate tolerance had been considerably increased, and, when later the insulin was gradually withdrawn, the patient remained free from glycosuria on the increased carbohydrate. Four months later this man was readmitted suffering from a carbuncle on the back. The urine contained much sugar and acetone ++++. On this occasion he left hospital with his carbohydrate allowance unchanged from the previous dismissal; but now he required 35 units of insulin per day to keep the urine sugar-free.

The comparatively poor results as regards increased carbohydrate tolerance seem to me to be explained very largely by the susceptibility of the diabetic to intercurrent infections, often slight in their nature. Thus a chill, a gumboil, a tonsillitis, - any one of these may interrupt progress and may even cause a reduction in carbohydrate

tolerance.<sup>33</sup> One case, a man of 34 (not included in this series as the records are incomplete) was treated by diet alone, and the carbohydrate level reached 90 gms. per day. This amount remained unchanged for 8 months, during which time the urine remained sugar-free and his weight stationary. In March 1924 he contracted influenza, and he was ill for 3 weeks. Sugar appeared in the urine in amount, and the patient subsequently required 16 units of insulin per day to prevent glycosuria on 70 gms. of carbohydrate.

Few patients escape entirely from some such rather trivial infection, and, with such a result as in this case before us, we can hardly wonder if in general the carbohydrate tolerance does not improve.

A C I D O S I S.

Speaking generally in the cases admitted with severe acidosis where death was averted, the condition gave rise to little anxiety after the first 48 hours. Indeed the rapidity with which acetone disappeared from the breath and from the urine when insulin was administered was most striking. In one case acetonuria persisted for 18 days, (Case In. 1) but this was quite exceptional.<sup>16</sup> Occasionally patients returning to report showed considerable acidosis, but errors in insulin dosage or in diet were always found to account for this condition; and, once these were corrected, acetonuria rapidly disappeared.

SEPSIS.

S E P S I S.

It had always been recognised that any infection exercised a very detrimental effect upon the diabetic patient, and that the presence of a septic focus was especially disturbing. Even under "Allen" treatment the effect was very marked, as Case P.1 Chart 3 well illustrates. It was confidently anticipated that insulin would very greatly lessen the gravity of this complication. The following 4 cases demonstrate its effect upon 2 patients on "Allen" treatment - a carbuncle in each case: upon two patients on insulin treatment - a purulent otitis media and an onychia: upon a case of glycosuria, subsequently proved to be a "Lag" in type, and suffering from an appendix abscess. The "Allen" cases :

(1) Case P.6. Chart No. 3. This patient had been under treatment 6 months before and had been discharged without insulin. He was readmitted with a very large carbuncle on the left shoulder which had been incised immediately before admission. He had lost 4 kilos since his previous dismissal, and now looked very thin and ill. The urine contained much sugar, and acetone ++++. With large doses of insulin and an adequate diet healing proceeded very satisfactorily, and undoubtedly more rapidly than under the old routine. While in hospital on this occasion patient gained 10 kilos.

Case In. 13. This patient was also a diabetic of some years' standing who was being treated on modified "Allen" lines.

He was admitted to a "Home" suffering from a carbuncle on the right cheek. He looked thin and ill, and the urine contained sugar and acetone in amount. With 35 units of insulin, spread over 3 days, healing took place with remarkable rapidity; and thereafter insulin was dropped and patient was treated on the usual dietetic lines.

### Insulin Cases:

(3) Case P.1. Chart 4. This patient, a diabetic of long-standing, had been on insulin treatment for three months, but he had been gradually losing flesh, apparently owing to a chronic purulent otitis media. This condition suddenly "flared up", and patient was admitted very ill, with very marked glycosuria, and with acetonuria ++. Before admission he had taken 100 gms. carbohydrate per day with 30 units of insulin. With increased feeding and insulin the ear condition improved steadily, and ultimately dried up completely in 4 weeks. While discharge from the ear was present the carbohydrate tolerance was much reduced, but subsequently it returned to its former level with the same dose of insulin - 30 units. He gained 2.5 kilos while in hospital.

In striking contrast to this result was the experience of the same patient before insulin was available. He was admitted in November, 1922, with a severe sepsis of the left hand, the result of striking his thumb with a hammer while at

work. With difficulty the hand was saved, and 2 months later healing was not complete. During this time the glycosuria could not be controlled by diet, and his general condition greatly deteriorated.

(Chart 3 is unsatisfactory towards its termination owing to some unsuccessful experiments in this patient to administer insulin by mouth along with glucose.)

(4) Case In. 50: This patient was under treatment with insulin, but difficulty was experienced in keeping the urine sugar-free, although sugar was never present in amount. Six weeks after admission, when progress appeared to be more satisfactory, patient developed an onychia. At once glycosuria became more marked, and, with a slightly decreased diet, insulin had to be advanced 15 units (from 20 to 35) to reduce the urinary sugar to a trace. The onychia healed rapidly and satisfactorily.

"Lag" Case:

(5) Case In. 33: This patient had been operated upon 2 days previously for an appendix abscess. Sugar and acetone were later discovered in the urine. Small doses of insulin were cautiously administered for 8 days with disappearance of acetone from the urine, and considerable improvement in the patient's general condition. Healing of the abscess was slow, but the surgeon was quite definite in his opinion that its progress was accelerated by the insulin treatment.

Two months later a Glucose Test on this patient showed

him to be a "Lag" case.

The effect of other infections is well seen in the case which I quote under "Results" , page 6 - a patient who developed an influenza and who required insulin as a result of his reduced carbohydrate tolerance.

From this small series of cases it seems clear that infections generally, and septic infections in particular, are not to-day to be so greatly feared as in the days before insulin was obtainable. Nevertheless, even when the patient is under treatment with insulin, they exercise a profound effect upon him, and he should be advised to take every precaution to avoid such complications whenever possible.



## HYPOLYCAEMIA.

If too large a dose of insulin be administered to any patient he experiences a train of symptoms known as a "hypoglycaemic reaction". The level to which the blood-sugar must be reduced in order to bring about this reaction varies in each individual,<sup>13</sup> but generally it is in the neighbourhood of 0.05%. Patients whose blood-sugar is high may experience the same symptoms when any marked reduction takes place, even although the level of blood-sugar may not have fallen below 0.10% (Leyton).<sup>34</sup> The symptoms complained of are hunger, a feeling of apprehension, shakiness, sweating, constriction in the throat, and tightness in the chest. If nothing is done to alleviate the condition delirium and convulsions may follow, and, as recorded in one case, death. In my series of cases I have never met with a severe reaction, nor have I seen one elsewhere. Four of the earlier cases (In. 1: In.2: P.2: P.5) experienced slight reactions, each complaining of marked hunger, "nervousness", sweating, and palpitation, but in each case prompt measures were taken, and the symptoms passed off in 3-5 minutes. In each case a cup of hot unsweetened tea with 10 gms. of white bread were all that was required. Glucose, either by mouth, by rectum, or intravenously has never been necessary, nor has adrenalin been used. In 2 cases the attack occurred at night, the patient waking up about 1 a.m. acutely uncomfortable; but

generally it occurred after the larger morning dose and immediately before the one o'clock meal. As I have stated above the smaller dose was always given in the evening, and, where insulin was being advanced more rapidly than usual, a small carbohydrate meal was given last thing at night. The important point, however, in seeking to avoid the discomfort or danger of hypoglycaemia is to advance the insulin slowly, controlling each addition to the dose by urinary tests, with blood-sugar tests whenever possible.

There is always the possibility that a patient on insulin - not necessarily during the stage of investigation, but at any subsequent injection - may receive his dose of insulin, and later find himself unable to eat the meal which should follow. In such a case patients are advised to eat the carbohydrate portion of the meal if possible, but, if this cannot be taken, then a corresponding amount of bread and milk or even of sugar (in grammes of carbohydrate) should be substituted. Very rarely has trouble of this nature been experienced, but it is always wise to give such instructions in case of need.<sup>35</sup>

All patients are ordered to carry sugar in "lump" form with them, and to have sugar beside them at night. Up to the present, as I have said, no patient has had to have recourse to this sugar so far as I know.

Poulton<sup>56</sup> has advocated that each patient should be made to experience a slight hypoglycaemic reaction before the stage

of investigation is complete, since, once the character of the symptoms is known, that patient has never any difficulty in recognising their onset again. Recently I have adopted this procedure more generally, but I am not convinced that it is a wise one. These patients tend very often to be unduly apprehensive, and to consume extra carbohydrate without reasonable provocation; but, as in many other forms of treatment, it is sound practice in the right type of case. On the whole, nervous patients are better not to be treated in this manner.

In 2 patients a reaction occurred at night, and the patient wakened up with the characteristic symptoms. This was a reassuring occurrence since there had always been a little anxiety lest the attack should occur at night, when the patient might pass from sleep into a convulsion or into coma without being able to give warning of his condition. With this fear assuaged it would seem that the dangers attached to hypoglycaemia have been much too strongly represented.<sup>25</sup> It is not denied that serious and alarming symptoms may occur as in cases recorded by Meakins,<sup>45</sup> Cammidge,<sup>12</sup> ~~Graham~~<sup>31</sup>, Joslin, and many others.<sup>46</sup> But these cases were all confined to the earlier days of insulin, and, since perhaps even greater care in its administration was being taken at that time, one cannot but wonder if the less perfect methods of manufacturing and standardising the drug at that time may not be held to account for them. From personal communications with

various workers I am satisfied that to-day hypoglycaemic reactions are less common, and less severe in type when they do occur. I wish to quote at some length 2 cases recently admitted to hospital in support of this latter contention.

Case I (Case P.8: a subsequent admission)

J.D. age 8, was dismissed from hospital on 26/7/24 very well. He was regularly at school until March 1925 when he began to "take fits". According to his mother's statement each fit occurred about 4 hours after the morning dose of insulin. It commenced with a twitching of the limbs and face, and the eyes "turned in". Thereafter the boy fell to the ground unconscious. The twitchings became general and more marked, and lasted for about one minute. The boy seemed to pass then into a natural sleep from which he awakened rather dazed, but otherwise apparently normal. He had four such attacks in all. It was then discovered that a larger dose of insulin than usual had been given on these four days (owing to a change in syringe) used for injection and the fits were put down to hypoglycaemia. About one month later he had another fit, but this time it seemed improbable that it was hypoglycaemic in origin. On 11/4/25 he had a severe convulsion at 5 p.m. (9 hours after the morning dose of insulin). The twitching affected the face, left arm, and left leg. Upon waking from the sleep which as usual followed he was found to be paralysed in the left arm and

leg. He complained of headache, and was observed to rub the right side of his head frequently. On 13/4/25 patient was admitted to hospital, he was looking and feeling very well. Nothing abnormal could be found on physical examination. The urine contained sugar 10 gms: no acetone.

Patient received C.60: P.45: F.100 with insulin pushed to 45 units in order to provoke a hypoglycaemic reaction. None occurred, however, and on 6/5/25 the insulin was reduced to 30 units. On 8/5/25 patient seemed rather drowsy in the morning, but it did not appear to Sister to be abnormal, and he received his morning insulin injection (15 units) as usual. He took a good breakfast, and appeared to sleep most of the forenoon. At 1.45 p.m. he was found to be very drowsy indeed and to have passed urine in bed. He lay upon his back, breathing heavily, with saliva trickling from his mouth. He could not be roused. All reflexes were present, but rather sluggish, and there was no evidence of paralysis of limbs. He was, however, unable to swallow. As hypoglycaemia was suspected patient was given 50 gms. glucose by rectum at 2.15 . At 2.30 p.m. he suddenly called out, and this cry was followed at once by a generalised convulsion. The face became very cyanosed and the tongue was bitten. The convulsion lasted about 1 minute. The body then became completely rigid, and this state persisted for 2 minutes. With relaxation consciousness returned to some extent. Babinski's sign was positive on

both sides. Patient slept for about half an hour and on waking up he appeared completely normal. The plantar reflexes were both flexor. Save for slight frontal headache he made no complaint. A blood sugar estimation at 4.30 p.m. gave a reading of 0.06%.

There were no further attacks during his residence.

Case 2: (Case P.2: subsequent admissions). T.D., aged 17, was admitted to hospital on 7/5/25. He had been very well until the evening of 3/5/25 when he walked out of the house in a state of partial undress and was brought home by a neighbour. He appeared very dazed, and did not answer questions. In bed that night he passed urine. Next morning he remembered nothing of what had happened. He complained only of headache and went to work as usual. That day (4/5/25) he had three attacks of left-sided twitching, the first occurring just before his one o'clock meal and the others in the afternoon. He fell to the ground in each, but did not lose consciousness. On 5/5/25 he was well all day. Late in the evening he took a fit in which he threw himself about, cried out, frothed at the mouth, and passed urine involuntarily. After an hour's interval he took another. Thereafter he slept quietly, but there was incontinence of urine through the night. On 6/5/25 he was very well. Each day he had had insulin 15+10 units, and he had eaten his usual diet at the meals taken at home. He was given nothing apart from the food at

meals.

He was admitted to hospital on 7/5/25. He appeared very well. The blood sugar on admission was 0.30%: urinary sugar, 35 gms. He was treated for two weeks on his previous diet and insulin dose, but proper treatment was impossible as he persistently broke diet. He had no fits or twitchings while in hospital.

On 9/7/25 he was readmitted. His mother stated that he "took fits every two or three days", generally about 10 p.m. or between 2 and 3 p.m. In these the limbs twitched, urine was passed, the tongue was bitten, and he was very noisy.

On 9/7/25 he was conscious but dull and apathetic, quite unlike his normal, confident self. The breath smelt of acetone. The urine contained sugar 3.1%: acetone +++: diacetic acid ++. He was given C.15: P.4: F.0 with 10 units of insulin. At 12.15 p.m. he was found to be very dazed, and shortly afterwards he screamed and struggled, and had to be held in bed. There was no incontinence, the tongue was not bitten, and he did not lose consciousness. Gradually he quietened down and complained of headache. Shortly afterwards he was sick and vomited. A blood-sugar at 2 p.m. was 0.30%.

On 10/7/25 (next day) patient received his morning dose of 15 units of insulin followed by a meal of C.20: P.5: F.0 at 8.30 a.m. At 10 a.m. the blood sugar was <sup>0.32%</sup>~~0.08%~~. At 12.15

p.m. he had a fit exactly similar to that described above, save that later consciousness was lost and the tongue was bitten. He fell asleep after the fit, and blood sugar readings were as follows :-

1 p.m.	-	0.082%.
2.30 p.m.	-	0.082%.
4 p.m.	-	0.115%.

Meal C.	P.	F.
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9 p.m.	-	0.302%.
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The evening dose of insulin was omitted.

During his residence there were no further attacks. Again treatment was difficult on account of misbehaviour.

These two cases are very similar. They might, of course, be epileptics, but the family history in both was good, and there was no history of previous fits in either lad. It seems much more probable that both were suffering from hypoglycaemia, in the first case due to the actual level of the blood-sugar (0.06% after the fit), in the second case due to the relative fall in blood-sugar level. (0.32% - 0.08%) Indeed the susceptibility to insulin in the second case is very striking, and contrasts strongly with his response on numerous occasions when in hospital previously. It also opens up interesting speculations regarding the effects of prolonged insulin administration. The point I wish to make, however, is that if these attacks were hypoglycaemic in nature, as seems more than likely, then recovery would appear to have occurred spontaneously on numerous occasions if the mother's stories



are to be credited. The recovery in case 2 in hospital without carbohydrate being given would certainly bear out the truth of their statements. It would thus appear that hypoglycaemic reactions, while alarming, are not necessarily attended by very grave consequences when left untreated; and while this fact would not excuse carelessness in the administration of insulin, or the neglect of precautions, it furnishes one with greater confidence in its use in adequate dosage in those difficult cases where the urine is free from sugar and blood-sugar estimations cannot be performed.

I N S U L I N   B Y   I N U N C T I O N .

In April 1923, in collaboration with Dr. Telfer,<sup>63</sup> an insulin ointment was prepared which, when rubbed into the skin of the rabbit, caused a fall in the blood-sugar. The ointment was prepared by rubbing up insulin in powder form with suitable bases (lanoline; paraffin. m<sub>ol</sub>l<sub>is</sub> ) in a mortar. The preparation was tried on various patients (P.2: P.4: P.6), but, although it was calculated that from 50 - 100 units were used at each inunction, no beneficial result was produced. Chart T.5 illustrates the effect of inunction (50 units) upon the blood sugar curve following a fixed meal, and contrasts the result with the effect produced by 10 units of insulin administered hypodermically. In man insulin by inunction would appear to exercise no influence upon the blood-sugar.

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No. 17.  
large injection of insulin which alone was available at that time, even over the abdomen. To-day, as the result of prolonged insulin treatment, the boy was running about the ward and in appearance compared not unfavourably with many of his fellows. His weight was now 32 kilos (5 st. 8 oz.), diet carbohydrate 70, protein 80, and fat 100, insulin dosage 30 units a day, and he was sugar- and acetone-free with a blood sugar of 0.15 per cent.

All results proved abundantly the great value of insulin in acutely ill patients. It would seem to hold out hope of cure in "early" cases and undoubtedly it alleviated the lot of the more chronic. Unfortunately the treatment was expensive, and for hospital cases the "Allen" treatment must remain for the present the most useful despite its somewhat unsatisfactory results. It was a doubtful kindness to a patient to assure him that a diet had been found for him which would keep him well and satisfied provided he obtained a drug which he was unable to afford and whose administration was outwith his capabilities. In conclusion, Dr. Rennie referred briefly to another chart, which illustrated one of some experiments which were made in the administration of insulin by inunction following Telfer's rabbit experiments recently recorded. The chart showed the blood-sugar curves obtained in a diabetic following a meal (a) without insulin, (b) with insulin injected in the usual way, (c) with insulin inunction. No reduction in blood sugar was found, a result confirmed on other occasions both with an ointment of crude insulin prepared by Dr. Telfer and similar to that used by him on rabbits and with other preparations. No dose of insulin which Dr. Rennie was able to give in that way—and he had endeavoured to give 100 units in one inunction—produced any effect upon the blood sugar. Insulin appeared not to be absorbed through the skin.

Dr. E. P. POULTON (London) spoke of the effect of insulin on relatively mild cases—those which became sugar-free on diet restriction. An attempt had been made to discover if any increase of tolerance took place after prolonged treatment. The rise of blood sugar was estimated after giving 25 grams of sugar, and the experiment was repeated after a period of treatment. The quantity of the diet was chosen so that in-patients felt satisfied; about 40 calories per kilo was allowed; the protein was about 1.5 grams per kilo, and during treatment the patients were walking about. After receiving 60 units a day for thirty days one case showed a gain in weight, but no evidence of improved tolerance. In one there was distinct improvement, in another perhaps a slight improvement; in one case after eighteen days' treatment there was a slight but definite improvement.

Dr. OTTO LEYTON (London) referred to the probable curative properties of insulin in diabetes mellitus of comparatively recent origin, and due to defect of the pancreas. Many had found that overworking the cells of the islets of Langerhans was followed by progressive degeneration. They had reason to think that whatever cause led to the sudden onset of diabetes mellitus, the destruction of the pancreas was, as a rule, not complete; some cells survived unchanged, some were destroyed, and some had their vitality reduced. Evidence of this consisted in the fact that a dog with a small fraction of pancreas, but of healthy pancreas, might be submitted to a general anaesthetic time after time without causing any recognizable deterioration of the pancreas, while the tolerance of the majority of diabetics was decreased by the administration of chloroform, ether, and sometimes of nitrous oxide. When there was a probability that damaged cells were present, it was thought worth while to attempt to nurse them back to health by rest. Rest of the pancreas was procured by giving sufficient insulin to maintain a slight hypoglycaemia. This might lead to symptoms of hypoglycaemia, but these were controlled by the administration of sugar. There was reason to believe that the tolerance of early cases improved. An attempt was being made at the London Hospital to obtain evidence by comparing the curve of the blood sugar while and after giving dextrose intravenously over a period of three hours, before and after a course of treatment.

Dr. A. P. THOMSON (Birmingham) said that he had treated over 30 cases of diabetes with insulin, and he had observed definite increase in tolerance after a period of treatment in two cases that had been previously rigorously dieted for a considerable time. He mentioned these cases specifically, as Dr. Banting himself seemed to doubt whether such a thing could occur. As the result of his experience Dr. Thomson believed that it was quite impossible to treat diabetic patients who could not learn to take care of themselves to a large extent or those patients who lacked the determination necessary for the limitation of their diets, unless, of course, continuous control could be arranged. Hospital practice was frequently disappointing. With reference to the value of blood-sugar estimation in the control of treatment, Dr. Thomson found that it was possible in the average case to get along with very few estimations during the preliminary period of standardization in a nursing home, and that afterwards he had not found them necessary providing the urine was examined frequently enough and that the diet was properly controlled. He believed that the liability to toxic symptoms after insulin decreased with the improvement in the condition of the patient. He mentioned as unusual complications of insulin treatment the occurrence of acute cataracts in a patient who was otherwise doing well and two instances of transient haematuria after large doses. He agreed with Dr. Leyton that the so-called hypoglycaemia crisis frequently occurred when the blood sugar was above the normal, and he believed that the sudden change in level was more important than the exact value.

Dr. F. A. ROPER (Exeter) detailed an incident in the case of a male patient, aged 54, whose general condition, blood-sugar curve after glucose meal, and acidosis estimations showed diabetes of a considerable severity. He had some few months ago had diabetic coma and there was considerable albuminuria. Starvation was badly borne, so that on the fourth day despite considerable glycosuria he was placed on a diet of about 600 calories containing 16 grams carbohydrate, while insulin 20 units per diem was commenced. On the third day of this diet the insulin was increased to 30 units given in three equal doses. The urine was closely watched for disappearance of sugar. On the fifth day the urine at 9 a.m. contained sugar, and the blood sugar on the day preceding was 0.20. Lunch was taken at 1.15 preceded by insulin. An hour later he expressed himself as feeling "very fit." A quarter of an hour later he was found comatose—unconscious—with deep abdominal breathing tending later to become irregular (Cheyne Stokes). From the first he was unable to swallow, and, glucose per rectum failing in any way to improve his condition, in fifteen minutes or so 2½ drachms of glucose in a pint of saline were administered intravenously. Ten minutes after commencing the injection he began to yawn, and in half an hour could speak and thereafter quickly recovered. A urinary specimen passed at noon that day was found to be sugar-free, this being the first time it had been sugar-free since he had come under observation. The incident was related (1) in support of the contention that at present all patients requiring insulin should be under hospital conditions of observation and treatment until their basal dietetic requirements and proper insulin dosage had been determined, and (2) as emphasizing the advisability during this initial period of frequent blood-sugar determinations inasmuch as hypoglycaemia in this case supervened within five hours of cessation of glycosuria with a moderate insulin dosage.

LIEUT.-COLONEL E. E. WATERS, I.M.S., asked Dr. Banting if the temperature at which insulin was stored had any effect on its potency; and Dr. CLARKE BEGG (Swansea) inquired what instructions should be given to patients on leaving hospital. Should they be sugar-free or discharged with a small degree of glycosuria?

#### Dr. Banting's Reply.

Dr. BANTING, in reply, said that the cause of deficiency of the islets of Langerhans was not known. Patients requiring continuous treatment were trained to give their own injections. Larger doses of insulin were always

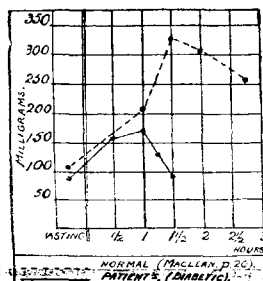
From the figures quoted it would be apparent that an improvement might be looked for in the great majority of cases treated by the "Allen" method, a result quite in accordance with that of all others by whom it had been employed. But while the immediate prognosis was good, experience had shown that in hospital patients the end-results were far from satisfactory. It was rare to find a patient of that class who did not lose ground after discharge from hospital. Almost all who had been readmitted had shown a distinct loss of weight and a decreased carbohydrate tolerance, not infrequently accompanied by thirst, polyuria, etc. Unwillingness to adhere to the diet was responsible in many cases; but an inability to understand the rationale in some and the conditions of life, particularly at the present day, in others made it very difficult, even for those who were most anxious to keep well. Among private patients the prognosis was, of course, much better. It should be remembered that it was necessary to insist upon these patients reporting for examination from time to time. It was not uncommon to find a zealous patient grossly under-feeding himself without regard to his general condition in an endeavour to remain sugar- and acetone-free at all costs. One patient, a woman of 35, who had been under treatment two years before with good results carried on unaided until six months ago, when a recent but persistent cough and spit again brought her under observation. She confessed to long periods of semi-starvation with deliberate loss of weight, but she was proud of the fact that she had remained sugar- and acetone-free. The general condition had greatly deteriorated and there was an obvious tuberculous lesion at the right apex.

3. *Insulin*.—Ten cases had been treated with insulin and the result in each case had been a distinct improvement. All save two were cases of severe prolonged diabetes. With one exception they had all previously been in the wards under "Allen" treatment, four of them on more than one occasion. It was originally intended to put each patient upon what had been his optimum diet when last dismissed from hospital, but all had retrogressed considerably and this procedure could not be followed. Four cases, however, commenced treatment in this way, but in each case it was found that there was a considerable reduction in the carbohydrate tolerance, much sugar in the urine, some acetone and a high blood sugar. All had lost weight. An effort, not always successful, was made to render each sugar-free by starvation methods and thereafter to work up to what was considered a sufficient diet, using insulin to keep the patient sugar-free. The method adopted in determining the requisite dose of insulin was, briefly, as follows. To each patient was given a meal of known carbohydrate, protein, and fat content, called a "fixed meal," with increasing doses of insulin, and this procedure was continued until satisfactory blood sugar and urinary sugar results were obtained. From the information thus obtained a sufficient dose for an adequate diet was calculated in each case. A chart showed the effect of such a meal—carbohydrate 20, protein 18, and fat 25—upon the blood and urinary sugar curves obtained by hourly estimations in (1) a normal individual, (2) a diabetic without insulin, (3) a diabetic with insulin, in that particular instance 10 units. The fall in the curves in (3) was very striking. This method of treatment was most suited to chronic cases with a low carbohydrate tolerance. These patients were kept under their best weight and the blood sugar tended to run above normal limits. Two patients, adjudged to be cases of comparatively recent origin, were treated on the usual "Allen" lines, but the carbohydrate tolerance was low and acetone readily appeared in the urine. Sufficient insulin was given to keep the patients sugar- and acetone-free with a normal morning blood sugar and at a reasonable weight. This method, on the lines of that described recently by Graham and Harris, seemed to be most satisfactory in "early" cases. Four cases showed so pronounced a degree of acidosis that insulin administration with abundant carbohydrate had to be commenced at once. In these patients the urinary sugar was entirely disregarded during the earlier treatment, attention being directed to the urinary acetone and diacetic acid, the alveolar  $\text{CO}_2$ , and the ammonia-nitrogen ratio. Large doses of insulin, 40 to 60 units in the day, were given, the results being controlled by blood-sugar estimations  $3\frac{1}{2}$  to 4 hours after injection.

In no case did any untoward symptoms follow such doses at this stage of treatment, and it would appear that so long as sugar and acetone were present in the urine the estimation of the blood sugar was unnecessary. When rendered acetone-free the treatment of these patients followed lines similar to that of those described above. All patients treated by insulin increased in weight, the gain varying from 1 to 8 kilograms. All showed a very definite improvement in general condition, mental as well as physical. In all save one case, sugar and acetone rapidly disappeared from the urine, the acetone sometimes with dramatic suddenness. The exception was a man admitted with marked acidosis, to whom insulin was given in large doses, but the urine was not rendered acetone-free for twenty-one days nor sugar-free for thirty-eight days. Attempts subsequently to reduce the dose of insulin in that case had always been followed by a return of sugar and acetone to the urine. He would appear to have no pancreatic tissue capable of recovery. A typical result of treatment was illustrated by another chart, which represented the later period of residence of a patient who on admission had shown 85 grams of sugar, much acetone and diacetic acid on a diet of carbohydrate 60, protein 25, and fat 5; he weighed 51 kilos. With an initial dose of 30 units of insulin and gradually decreasing doses he was kept sugar-free and acetone-free for twenty-two days upon carbohydrate 90, protein 80, and fat 100. During that time the blood sugar fell from 0.35 per cent. on admission to 0.10 per cent. fourteen days later, and thereafter the pre-breakfast blood sugar remained within normal limits. The dose of insulin was reduced to a single morning dose of 5 units upon an unchanged diet, but at that point the patient began to add unauthorized carbohydrate to his diet, and sugar appeared in the urine. Home circumstances compelled him to leave hospital, but subsequently he was able to continue on a diet of carbohydrate 80, protein 80, and fat 100, with a weekly starve day, without insulin, sugar- and acetone-free. His weight on dismissal was 52 kilos. The chart showed an undoubted increase in carbohydrate tolerance as the result of insulin treatment. It might be argued that a similar result could have been obtained in this case by "Allen" treatment alone, but on previous admissions the patient had shown a decreasing carbohydrate tolerance and a general downward tendency. So satisfactory a result would have been most improbable.

The effect of insulin had been to allow restoration of pancreatic function, but this would seem to occur not only in cases treated on low diet and with a normal blood-sugar level, but also in those who for one reason or another were given a full diet with larger doses of insulin and in whom the blood sugar was above normal. He had seen this strikingly illustrated in one private case so treated in which symptoms of hypoglycaemia had led to a gradual reduction of insulin dosage without increased urinary sugar and with very considerable general improvement. The morning blood sugar in this case was 0.18 to 0.22 per cent. and the diet had remained unchanged. A third chart contrasted the results of "Allen" and insulin treatment in a telegraph boy, aged 13, first admitted three years before. The acetone, blood sugar, urinary sugar, weight, and dismissal diet were charted in that order. It would be seen that the first period of treatment was very satisfactory. The second period, two years later, was not so good, although the urine was rendered sugar- and acetone-free and the patient gained slightly in weight; but the dismissal weight did not equal that of two years previously in a growing boy. The carbohydrate tolerance, too, had considerably decreased. His third stay in hospital six months later was entirely unsatisfactory. It was now found impossible to prevent his adding to his diet, and he was dismissed irregularly with acetone and sugar in the urine and his carbohydrate tolerance never discovered. The blood sugar, however, was brought below 0.15 per cent. In March of this year the patient was readmitted to hospital. He was now 16 years old and terribly emaciated, weighing only 24.5 kilos (3 st. 11 lb.). The blood sugar was 0.5 per cent. The urine contained acetone and diacetic acid and was loaded with sugar. He was incapable of moving himself in bed, all the reflexes were absent, and the skin was so tightly stretched over the bones that great difficulty was experienced in administering the comparatively

five cases of diabetes with insulin, and allowing for a large measure of enthusiasm it had appeared to produce almost magical effects. Dr. Banting seemed to have already answered one of his questions: Was a patient any better off for being sugar-free if the blood sugar remained considerably above normal? Such a patient might be metabolizing more carbohydrate and gaining in weight and well-being, but with hyperglycaemia was he safe? Possibly if it had been ascertained that glycosuria occurred with a low blood-sugar threshold, they could make use of a trace of glycosuria as the guide to insulin dosage. Dr. Banting had advocated Woodyatt's "basal requirement diet." Dr. Nixon had found that a most practical standard diet. It enabled them to adjust the insulin dosage to a diet which was adequate. Moreover, Woodyatt's formula for calculating the proportion of fat to carbohydrate and protein had proved most useful. As an example, a woman with glycosuria on whom the surgeon wished to perform myomectomy was rendered sugar-free in the urine by diet, but had a high degree of acetoneuria. The diet was recalculated, so that the fat was equivalent to twice the carbohydrate *plus* half the protein. In three days she was acetone-free and the operation was successfully performed. He had had an example of the personal factor in tolerance of insulin. A man, aged 29, was rendered sugar-free (in the urine) by complete starvation, but he developed glycosuria on a diet containing only 300 calories. He was given 5 units of insulin during starvation and he was not given a meal until one



Blood sugar after 50 grams of glucose by mouth.

and a half hours later. Before this meal his blood sugar was again estimated. His fasting blood sugar was 0.3 per cent. One and a half hours after insulin, while still starving, his blood sugar was 0.29 per cent. This man was ultimately enabled to take a diet containing 1,679 calories with a daily dosage of 40 units of insulin injected as 20 units twice a day. The symptoms due to overdosage of insulin were difficult to determine. A man of 40 receiving 20 units of insulin daily for three days developed sudden partial aphasia; he was given glucose before his blood sugar could be estimated. By the next day his aphasia had practically recovered; but this patient had previously shown symptoms of angina pectoris. This case rather frightened him, but he was not convinced that his aphasia was due to insulin. Another man, aged 35, receiving 20 units of insulin daily, told him that since the injections his feet suddenly become swollen at night and he felt tinglings in his legs; but this patient's blood did not show hypoglycaemia. A girl, aged 14, presented an example of the difficulty occasionally experienced in deciding whether a patient was a true diabetic. Five weeks before she came under observation she complained of thirst, polyuria, weakness, wasting, and glycosuria. She was put on a diet before her blood sugar was estimated. The glycosuria disappeared and her blood sugar was no more than 0.13 per cent. In hospital her blood sugar fell to 0.8 and 0.75 per cent., although she was supposed to be taking 7 ounces of bread a day. She had also ketonuria. She was tested with 50 grams of glucose, given by mouth, and her blood-sugar curve was characteristic of true diabetes. Then she admitted that she had been afraid to eat the bread permitted in her diet. Ultimately she was found able to take a diet containing 3,000 calories; her blood sugar remained about 0.1 per cent., and she had been sent home as not needing insulin treatment. She was a potential diabetic with a high sugar tolerance. An excessive indulgence in chocolate had produced her first symptoms of diabetes.

Dr. J. K. RENNIE (Glasgow) said that the treatment of diabetes had undergone so marked a change during the past few years that a consideration of the results obtained under the various methods of treatment was not without interest. The following figures were obtained from the records of admissions to Dr. Cowan's wards in the Glasgow

Royal Infirmary, and covered a period of thirteen years. For convenience he had divided the cases into (1) old, (2) Allen, (3) insulin, the terms applying to the type of treatment adopted in each group. So far as possible only cases of undoubted diabetes had been included, but as all the "old" and a few of the "Allen" cases were under treatment before the routine use of blood sugar and glucose tests the records in these groups were necessarily less trustworthy. Needless to say, information had been received from time to time of the subsequent death of many of these patients, but as much of that information was unreliable it had been disregarded. Only cases which died in hospital were included in the following table:

	Patients Treated.	Improved.	Stationary or Worse.	Died.	Mortality.
1. Old ...	49	29	12	8	16.3%
2. Allen ...	47	39	6	2	4.2%
3. Insulin ...	10	10			

1. "Old" Cases.—This group included all cases treated under the various dietetic régimes in vogue before the introduction of the well known starvation methods of Allen and of Graham. A reference to the ages of the "improved" group showed that 11 cases were under 35 years of age, while in the stationary group 9 of the 12 cases were under that age, and 3 of these under 20. Of the deaths all 8 occurred in coma; 7 of these patients were under 35, 4 under 20.

2. "Allen" Cases.—Forty-seven cases were treated on "Allen" lines, and an improvement, more or less marked, occurred in 39. In this group, however, the average age was rather less than in the corresponding group of "old" cases, 17 being under 35 years. Of the 6 cases in the stationary group 3 became dissatisfied with restricted diet and left hospital irregularly, a happening which was becoming increasingly less common—probably the only good result of the publicity which the treatment of this condition had received of late in the public press. One case had a progressive pulmonary tuberculosis on admission and went to a sanatorium; and one, a girl of 17, suffered from mitral disease with failing compensation. The sixth case, an elderly man with long-standing diabetes and gangrene of the toes, made no progress under treatment. Of the two patients who died, one was admitted with air hunger, and died in coma in thirty-six hours. The second case was that of a youth of 17 in whom symptoms had first manifested themselves five weeks prior to admission. He was poorly nourished, but bright and alert, and nothing abnormal was found on physical examination. The urine contained sugar in amount, acetone was present, but there was no reaction with tinct. ferri perchlor. A liberal carbohydrate diet was ordered, but he had little appetite and took only about carbohydrate 30, protein 36, and fat 50 in twenty-four hours. Thereafter he became somewhat restless and drowsy. Thirty-six hours after admission he suddenly collapsed, the pulse becoming imperceptible. He recovered slightly under stimulation, but improvement was transient and he died ten hours later. At no time was there any suggestion of air hunger, the breathing throughout being easy, regular, and natural. A positive tinct. ferri perchlor. reaction was never obtained in any sample of urine. Another case seen more recently, a man aged 50, gave a history of having taken little food for three weeks prior to coming under observation. The urine contained much sugar but no acetone or diacetic acid; the blood sugar was over 0.4 per cent. There was some twitching of the muscles of the neck and upper chest and the breathing was slightly Cheyne Stokes in type. The pulse was rapid and of poor quality. Despite stimulation, liberal carbohydrate, and insulin in large doses he died, apparently of cardiac failure, thirty-six hours later. Cardiac failure was certainly an important factor in these cases and was a complication for which one must watch in all severe diabetics. Two of the cases admitted for insulin treatment showed signs of cardiac weakness, so that the routine "starvation" diet until sugar-free had to be stopped and replaced by feeding *plus* insulin.

repeated at fifteen-minute intervals for the first hour and subsequently at the end of each hour, for four or five hours, and the results were plotted out in a graph. In a healthy individual and in certain forms of glycosuria, the percentage of sugar and the hydrolysis value of the blood were practically the same, the difference value never exceeding 0.01 per cent.; but in ordinary cases of diabetes the difference value curve was much higher, either fasting or at some period after the meal. By comparing the curves from a large number of cases with those exhibited by animals in which hyperglycaemia had been experimentally induced by various procedures, it had been possible to divide them into three main groups: (1) reversed curves, in which the difference value was abnormally high fasting but fell as the percentage of sugar in the blood increased after the meal, to rise again as it diminished later; (2) concurrent curves, where the rise and fall in the blood sugar after the meal were associated with a corresponding rise and fall in the difference value, starting from a normal fasting level; (3) unrelated curves, in which the difference value was normal in the fasting state, but rose to an unusual level several hours after the test meal, generally as the percentage of sugar is falling. The first type was characteristic of deficiency of the internal secretion of the pancreas, the second was met with where there was hyperactivity of the thyroid, pituitary, or other ductless glands, and also in disturbances of the nervous system; the third group embraced cases where defects in the storage powers of the tissues, and particularly of the liver, for carbohydrate existed. Various combinations of these types were met with, and particularly a combined concurrent or unrelated curve with a pancreatic curve, for it would seem that deficiency of the internal secretion of the pancreas eventually developed in all untreated cases of persistent hyperglycaemia, whatever the initial cause might be. Taking a consecutive series of 250 cases it was found that there was a pancreatic factor in 202, or 80.8 per cent., but that in only 114, or 45.6 per cent., was pancreatic deficiency the sole cause of the glycosuria and hyperglycaemia; in another 69, or 27.6 per cent., the liver was apparently mainly at fault, and in 61, or 24.4 per cent., simple concurrent curves were found; in addition, there were 6 cases (2.4 per cent.) of "renal glycosurias" showing normal relations between the curves.

These findings were of more than theoretical interest; they had an important bearing on treatment, for it was well known that the Allen fasting treatment was based upon experiments with depancreatized dogs, and the benefits arising from the use of insulin in diabetes were due to the supply of the internal secretion of the pancreas introduced into the circulation at each injection. So long, therefore, as there was a pancreatic element in the condition they should expect improvement to result from fasting or insulin, and if the pancreatic deficiency was due to functional and not organic changes in the gland permanent benefit would follow; but if the hyperglycaemia was associated with advanced destruction of the elements forming the internal secretion of the pancreas or was entirely dependent upon non-pancreatic causes permanent benefit would not be induced either by fasting or insulin, however long continued. His experience with the fasting treatment tended to support these conclusions, and, although it was as yet too early to make definite statements regarding insulin, it seemed probable that they were equally applicable.

Insulin might be used in the treatment of diabetes in three ways: (1) as a temporary aid to metabolism in emergencies, (2) to enable the patient to take a diet permanently in excess of his natural tolerance, (3) as a remedial measure. In the treatment of diabetic coma, in the control of serious acidosis, and as a prophylactic in surgical procedures, insulin was of inestimable value. It was also of great assistance as a permanent aid to the defective metabolic power of patients who without its help were unable to take a bare maintenance diet; but its use merely in order that a luxury diet might be indulged in, as was now very commonly the case, was both wasteful and unscientific. Excepting in emergencies and in special circumstances insulin should be employed solely as a remedial agent and only in properly selected cases and under carefully controlled conditions. By means of the diagnostic methods to which he had referred it

was possible to determine whether a particular patient was, or was not, likely to benefit permanently from the treatment, and so to avoid unnecessary expense and the risk of failure with all its consequences in unsuitable cases. Essentially treatment with insulin was a means by which the effects of fasting might be obtained without the discomforts and dangers attendant upon prolonged abstinence from food, the injections enabling the work of the pancreas to be carried out vicariously and thus providing the physiological rest required for the recovery of its functions. Obviously, therefore, it was best employed in conjunction with the fasting treatment, or some modification of it, as Graham and Harris (*Lancet*, 1923, 204, p. 1150) had suggested. A permanent improvement of the food tolerance was only to be expected in those cases where experience had shown the fasting treatment to be of permanent benefit—that was, mainly in children and young people, where purely functional deficiency of the internal secretion of the pancreas was most commonly met with. Insulin was not a universal cure for diabetes, and he agreed with Joslin when he said (*Journ. Amer. Med. Assoc.*, 1923, 80, p. 1581) that "it is cruel for prominent individuals to make such a statement and arouse false hopes." In his opinion it was the most valuable means at our disposal for controlling defects of metabolism due to deficiency of the internal secretion of the pancreas, but it should not be looked upon as an empirical remedy to be given indiscriminately to all cases at present generally classified as diabetes. When its limitations and dangers, as well as its sphere of usefulness, were realized it would no doubt take its true place as a powerful aid in helping the recovery of a special form of metabolic defect.

Dr. GEORGE GRAHAM (London) spoke of results of experiments on patients in the medical professorial clinic of St. Bartholomew's Hospital. One patient, a severe type, who was always passing sugar in spite of starvation, was put on a diet of protein and fat up to 1,360 calories with 16 grams of sugar in the form of vegetables. In twelve days the urine was sugar-free and the morning blood sugar had fallen to 0.20. After six weeks the blood sugar was normal and remained so, although the calorie value was increased to 2,000 with additional protein and fat, and 80 grams of bread were tolerated. So far 20 cases had been treated on these lines; in 17 cases the blood sugar had fallen to normal, while in 3 other cases the blood sugar, although lower than before, had not yet fallen to normal. It was important to keep the blood sugar within normal limits, as Allen's experiments on diabetic dogs had shown that overworking of the  $\beta$  cells of the islands of Langerhans by doses of sugar caused degeneration of these cells. The aim should be to keep the blood sugar normal. The blood sugar should fall below 0.08 per cent. each day so as to rest the pancreas as much as possible. The results in cases of coma were so far good, two cases out of three having recovered; the third, who had a large abscess of the parotid, died three hours later in spite of 30 units of insulin; sepsis should always be thought of, as another case of coma had an acute otitis media. The dose of insulin varied in different patients; some would respond rapidly to 10 to 15 units, whereas others would need 40 to 60 units, and this could only be determined by cautious trial. Blood-sugar determinations were of assistance, but were not absolutely necessary. So long as sugar was present in the urine blood sugars could be dispensed with, but when the urine was sugar-free it was important to make estimation of the blood sugar occasionally as there was no other means of telling whether the blood sugar was 0.10 or 0.18 per cent. For blood-sugar estimations at this stage the blood should be collected either immediately before the insulin injection or about twelve hours after the last one. It was better to estimate the blood sugar as soon as possible, but the blood might be collected and preserved by the addition of potassium oxalate and one drop of 40 per cent. formalin per 5 c.cm.

Dr. J. A. NIXON (Bristol) said that it was a memorable occasion, which would mark the Portsmouth meeting in the history of the British Medical Association. They would be able to say, "I heard Dr. Banting's address on insulin." He proposed to take advantage of his presence to get enlightenment for his own ignorance. He had treated only

C. BY DR. J. K. RENNIE.

The type of blood-sugar curve obtained in cases of diabetes and in renal glycosuria after the injection of a known quantity of glucose has already been described to you. I wish briefly to describe the blood-sugar curve in cases of severe diabetes in

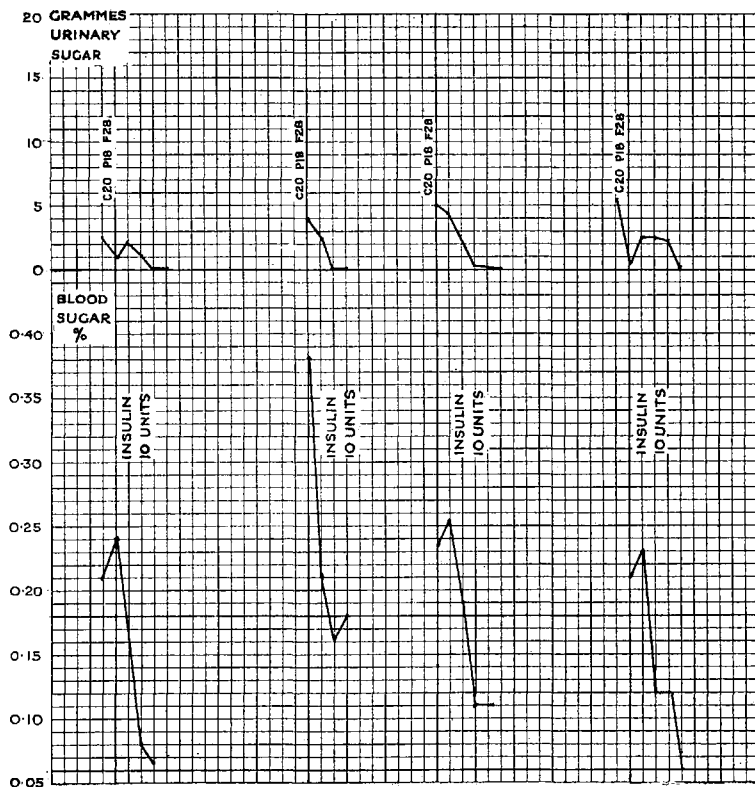


CHART I.

which insulin has been administered, and to draw attention to the effect produced upon the urinary sugar.

I should like, in the first place, to say a few words concerning the dosage of insulin. In the charts which I show to-night the dose is marked in units. The unit originally adopted was that amount of insulin required to lower the blood-sugar of a 1 kilo. rabbit to 0.045 per cent, at which level convulsions generally

occur; and this standard is in general use. It was found

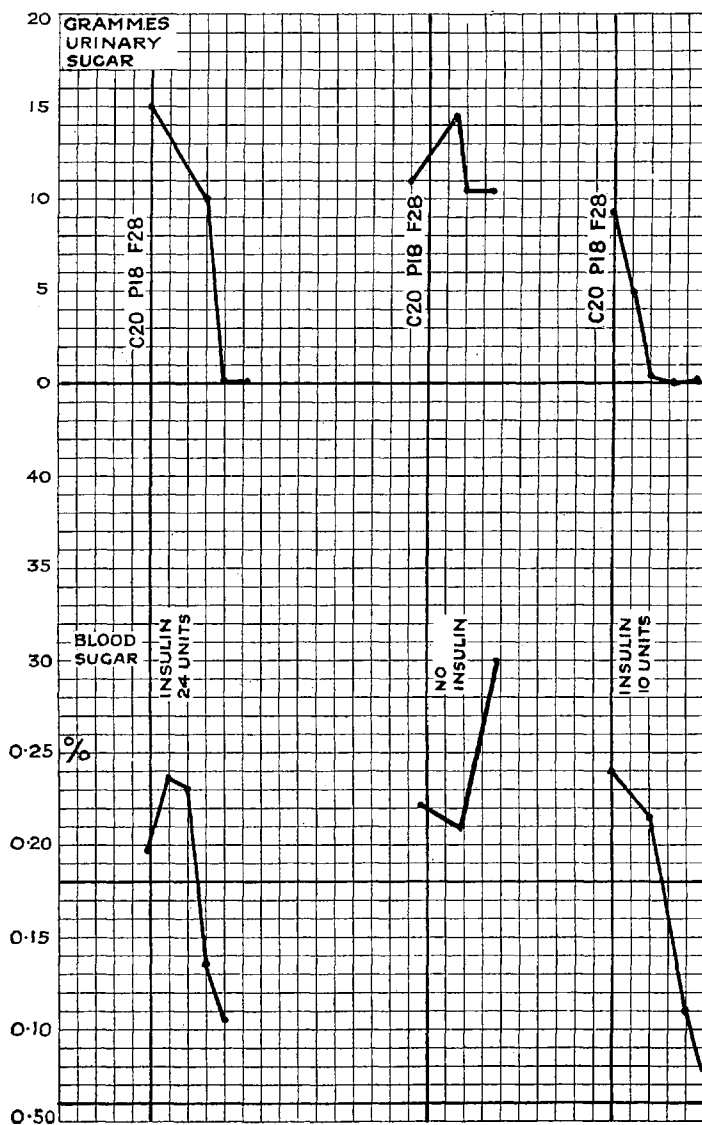


CHART II.

convenient to work with multiples of 5 units.

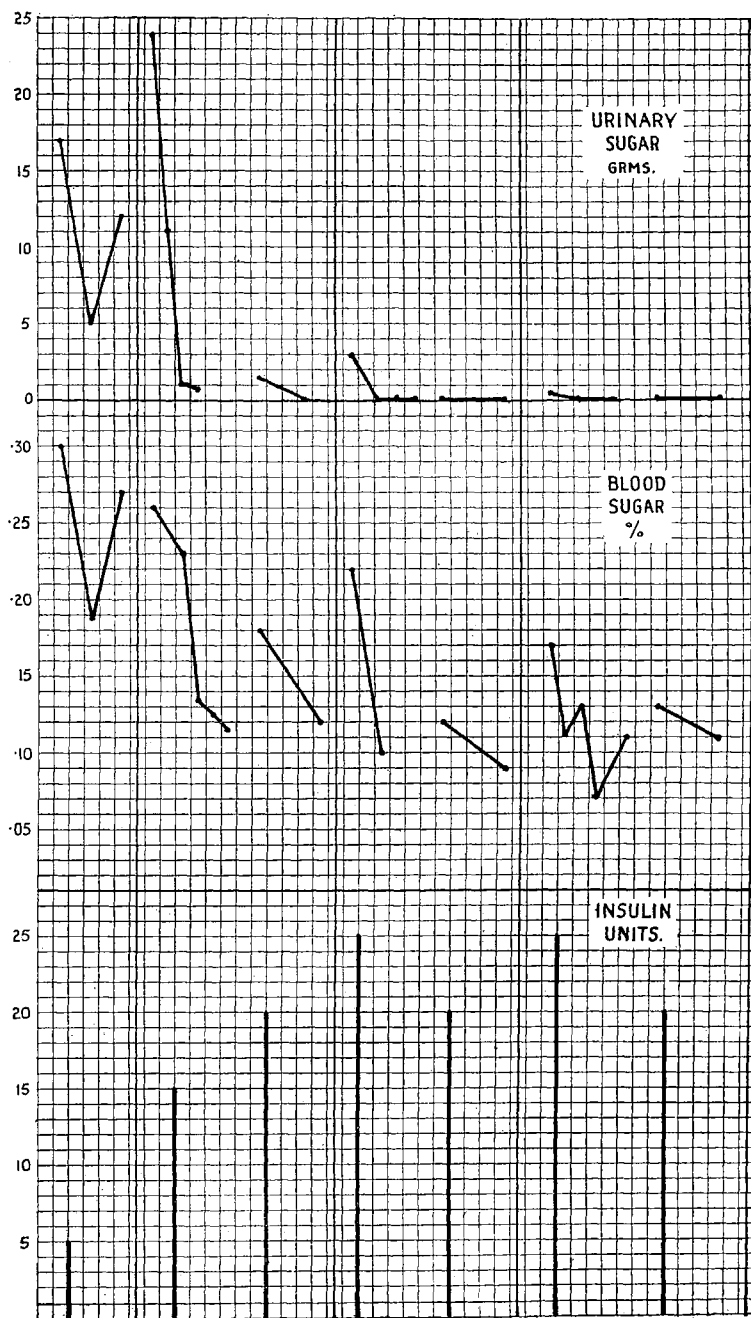


CHART III.



Our earlier supplies were obtained from the Physiology Department at the University of Glasgow, but, later, we used insulin from various manufacturers. As samples of insulin may differ in their potency, it was necessary to test each fresh supply before administering large doses. To do this we employed what we call a "fixed meal," *i.e.*, a meal where carbohydrate, protein, and fat contents were known, and which consisted of food-stuffs common to the diets of almost all our patients. Such a routine meal was found suitable in practically all cases, but under particular circumstances this procedure was modified, as, for

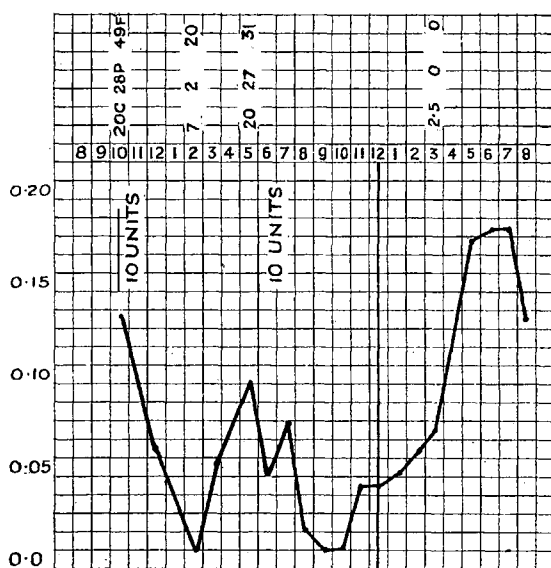
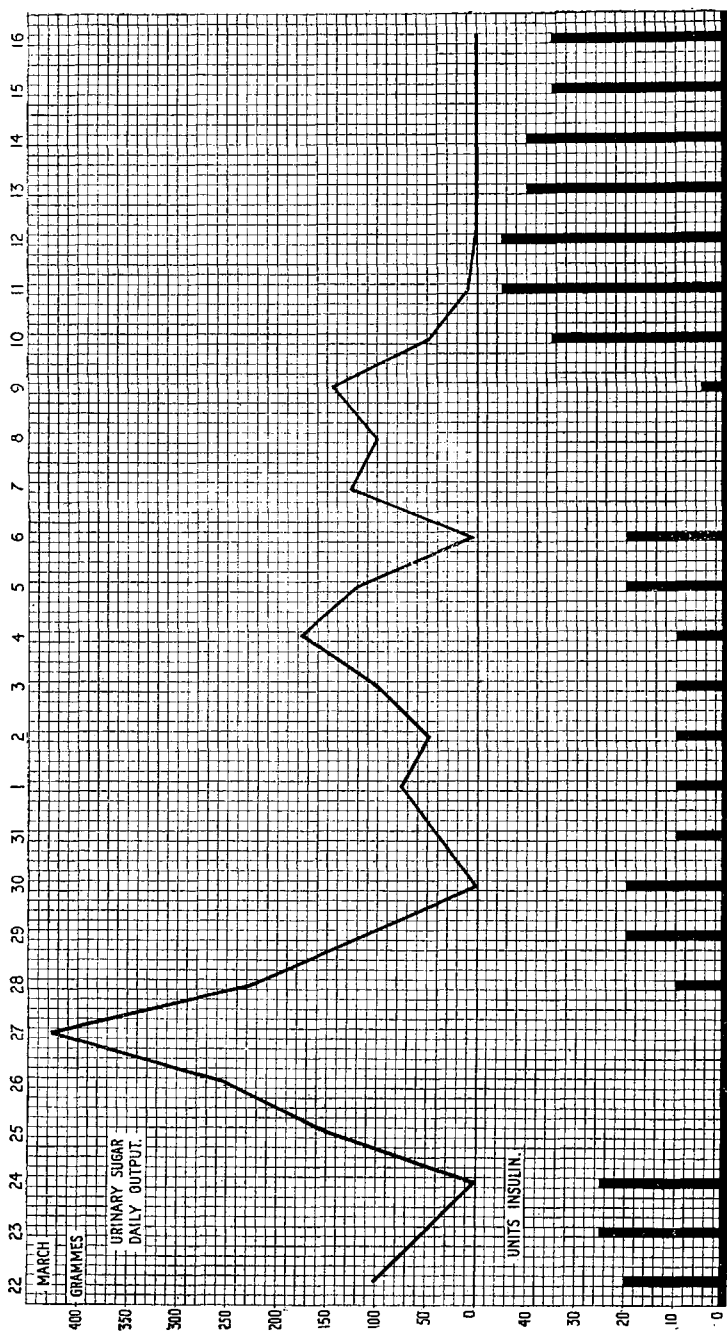


CHART IV.

example, in patients with severe acidosis. By this means we obtained information concerning the effect of insulin upon the blood-sugar and urinary sugar following a typical meal, and from these data we were able to calculate fairly accurately the amount of insulin which would be required when the patient was put upon an adequate diet. Needless to say, slight adjustments were necessary, but experience has shown that the main principle holds good.

The detailed procedure was as follows:—In the morning before any food had been given, or following a four-hours' starve, the blood and urinary sugar were estimated. This was



followed by a dose of insulin. Fifteen minutes later a "fixed meal" was given, and thereafter hourly blood and urinary sugars estimations were made for four hours. Chart I shows a series of typical curves obtained by this method, the "fixed meal" in each case consisting of C. 20, P. 18, F. 25, and the dose of insulin being 10 units. A rapid fall in blood-sugar from the second to the fourth hour is seen, while *pari passu* with this the urine becomes sugar-free. Chart II contrasts the results obtained from a "fixed meal" with insulin, with the blood and urinary sugar readings following a similar meal in the same patient without insulin. The rise in both curves in the case of the non-insulin meal is very striking.

Chart III shows the effect of increasing doses of insulin. It will be seen that the effect of 5 units of insulin is practically negligible, whereas with a morning dose of 25 units and an evening dose of 20 units the blood-sugar curve runs within normal limits, while the urine is sugar-free.

The reason for the larger morning dose will become quite apparent on examining Chart IV. This shows the blood-sugar curve in a diabetic on insulin where hourly estimations have been made over a period of twenty-four hours. The effect of the two doses of insulin on the blood-sugar is clearly demonstrated; but seven hours after the evening dose it will be seen that the curve commences to rise rapidly, finally reaching its maximum just before the next (morning) injection is given.

Chart V shows the effect of insulin upon the urinary sugar. This was one of our earlier cases when the supply of insulin was rather uncertain. The small earlier dose holds the sugar in check, but during two periods of three days no insulin was obtainable and at these points the curve rises sharply. Later on an adequate dose could be given daily. The urine becomes rapidly sugar-free and remains so.

In conclusion, I would remark that the routine morning and evening hypodermic injection is an obvious disadvantage in treatment by insulin. Recently we have tried an ointment containing crude insulin as an alternative method of administration. No difficulty has been experienced in rubbing this in, nor has any injurious effect been produced upon the skin. The results have been encouraging, and we hope to make further investigations along these lines.

On 27th September he collapsed, complaining of pain in the epigastrium and of great breathlessness.

On admission to hospital he was collapsed and cold. His pulse was frequent, small, and soft. The respirations were frequent, full, and sighing, and the breath smelt strongly of acetone. He was rather drowsy, but answered questions quite intelligently, though somewhat slowly. Under stimulation he improved, but at 17:00 he vomited, and his pulse at once failed. The sickness recurred, and at 22:00 he was pulseless and he died soon after midnight.

He was emaciated, but the skin was smooth and soft. Under insulin he became more intelligent even after his pulse began to fail, and the drowsiness practically disappeared. His death was due to cardiac failure and not to coma.

B. BY DR. J. K. RENNIE.

I wish to draw attention to-night to one or two points which the past year's experience of treatment with insulin has shown to be of importance.

In the first place, I would refer to the selection of cases for insulin. It is, of course, essential to differentiate between the renal and kindred types of glycosuria and the true diabetic. "Renal" cases are not very common, but they do occur, and they can be separated from cases of mild diabetes only by the "glucose test," with the details of which you are all familiar. By no method of urinary examination, with or without glucose, can this distinction be made with certainty, and as the patient's diet—and therefore his future comfort—depend on this finding, the test should invariably be performed.

Strict attention to diet remains the essential part of treatment even with insulin, and all patients must be taught to weigh their food. In practice this does not prove nearly so irksome as would at first appear. Within a very short time the patient is able to dispense with his scales almost entirely, and can tell with his eye the weight of any given quantity of the commoner foodstuffs. A periodic return to strict weighing should be insisted upon, particularly with regard to bread and the higher value carbohydrate foods, otherwise considerable errors

tend to creep in. Success in any but the mildest diabetics is impossible unless the food is weighed.

All diabetics should be treated in the first instance on "Allen" lines, or on some modification of this method. All cases admitted for the first time during the past year have been treated in this way, unless their condition necessitated the immediate use of insulin either through acidosis or other cause.

The earlier cases were treated by the older method—running out the fat from the diet, then the carbohydrate, starving till sugar free, adding carbohydrate gradually, and, later, protein and fat; but we have never been so cautious in the addition of protein and fat as was generally recommended, our object being to make the patient's stay in hospital as brief as possible. Recent work by Newburgh and Marsh has led to a general modification of diets on similar lines, but the amounts of protein are lower and of fat higher than we had employed. Our later cases, however, were treated with diets in which a similar adjustment had been made.

Almost all diets have conformed to the Woodyatt formula ( $F=2c+\frac{p}{2}$ ), though not infrequently diets containing much larger quantities of fat have been given with impunity while patients were under observation in hospital; but it is unwise to send a patient out on such a diet when it is unlikely that he will return regularly to report progress. For such patients insulin is safer treatment.

Reference has been made to the exceptions to the rule of "Allen" commencement. Patients who showed considerable acidosis received insulin at once. Twenty to thirty units of insulin is a perfectly safe initial dose in such cases. Fluids should be given *ad lib*—water, tea, coffee, clear soup. Opinions differ as to whether sugar should be given in these acid cases or not, but, as the blood sugar is already very high, it seems not only unnecessary but actually harmful. While generally agreeing with this view, Joslin allows small amounts. The insulin may be repeated at a shorter or longer interval, according to the severity of the case. A careful watch must be kept upon the patient, as it is quite possible that he might pass from the coma of acidosis into the unconsciousness

of hypoglycæmia. Wherever possible, frequent blood sugar estimations should be performed.

Insulin is frequently of value in expediting the treatment of cases which would probably do quite well on diet alone, but where acetone and sugar are difficult to clear from the urine. Under small doses both rapidly disappear, and thus a prolonged period of under-nutrition is avoided. The insulin is then discontinued, and subsequent treatment proceeds on the usual dietetic lines.

The other group of cases which require insulin on admission are those which show marked emaciation, frequently with signs of cardiac failure. Under-nutrition diets are contra-indicated in these cases, and a reasonable diet, allowing about 25 calories per kilo of body weight, should be given with insulin, cautiously added until sugar disappears from the urine. The blood sugar should, if possible, be kept within normal limits, but this is not always practicable. This line of treatment is generally indicated in long-standing cases where there is little, if any, recovery to be looked for, and where, leaving out the question of cardiac failure, a very strict regime merely makes the patient more uncomfortable without any reasonable prospect of benefit to follow; but we must recollect that eye changes, neuritis, pruritis, &c., occur more readily in hyperglycæmic patients, and it should be our object, by the more careful use of insulin, to retard the onset of these complications as far as possible. In other words, if you can keep the blood sugar low in these cases, do so; but do not lose your patient through inanition and cardiac failure in the process.

Almost all are agreed that, particularly in diabetes occurring in young people and in early cases, every endeavour should be made to keep the blood sugar within normal limits during the twenty-four hours (with a minimum of carbohydrate and of insulin). Particular attention is paid to a normal pre-breakfast blood sugar, the estimation of which alone is performed in cases under observation for a prolonged period. Fortunately, in young and early cases, the blood sugar is more readily controlled, and the great majority can be so treated. Unfortunately, however, most hospital patients are suited only for more or less heavy work, and they find their diets on these lines inadequate.

Our experience has been that they will eat more because their work demands more, and when that occurs they will eat what is most pleasant—carbohydrate. Probably under such circumstances more carbohydrate with large doses of insulin is better treatment.

Progress in patients treated with insulin can be controlled by urinary analysis alone. Some advise that a mere trace of sugar be allowed to appear in the specimen of urine passed three and a half to four hours after food, and certainly while this condition persists the patient is perfectly safe; but it is better treatment to push the insulin further to render the urine sugar free, though one is then in the dark as regards the level of the blood sugar, and an estimation is very helpful and comforting. Poulton recommends that insulin should be pushed until hypoglycæmic symptoms appear, the patient having previously been warned concerning what to expect. Obviously this procedure can be followed only in sensible and intelligent patients, and is not entirely without risk; but the patient, having once experienced a hypoglycæmic reaction, is never in doubt again about its onset. Nervous patients are apt to become alarmed when insistence is laid on the noting of these symptoms, and tend to devour sugar on little or no provocation. In these cases blood sugars are essential.

This "hypoglycæmic method" we have come to use more often, particularly when dealing with intelligent patients who have been under dietetic treatment for some time, and who have learned to test their own urine. We have had trouble at night on three occasions, and in each case the patient awakened up bathed in perspiration, shaky with palpitation, and very hungry. A cup of sweet tea soon restored them to normal. It is conceivable, of course, that they might have passed from sleep into a profound hypoglycæmic condition, but this did not occur, nor can I find any record of its having ever occurred. Trouble can be avoided by giving no dose later than, say, 4 to 5 o'clock, or by giving a small portion of the day's carbohydrate ration just before going to bed. This would appear to be a perfectly sound and safe procedure.

The dose of insulin should be kept as small as possible, partly because it is expensive and also because there would appear

to be an optimum dose for a given diet, beyond which much of the insulin injected would seem to be inactive. Joslin has brought forward evidence in proof of this statement. It is generally unwise to go beyond 40 to 50 units in the day. If a bigger dose should seem to be necessary, the diet should be very carefully scrutinised. Frequently, as McLean has pointed out, persistence with a large but apparently inadequate dose will lead to the patient becoming sugar free, and ultimately to a smaller dosage, the diet remaining unchanged throughout.

## II.—SOME POINTS IN THE ETIOLOGY OF DIABETES.

By DR. DOROTHY B. THOMSON.

In considering the question of the etiology of diabetes, there is one clue, the importance of which cannot be ignored, and that is the well-known fact that in diabetic patients the incidence of a septic condition invariably leads to an increase in the severity of the symptoms, shown by increase or recurrence of glycosuria, a rise in the blood sugar, and the appearance or increase of acetone and diacetic acid in the urine. This is well illustrated by the following two cases:—

1. The first, a man of 41 years, who had been under treatment for diabetes in the wards, was dismissed sugar free on 10th June, 1921. He kept well till 28th November, 1922, when he was readmitted with sepsis of the hand, resulting from a blow with a hammer. He was then passing 800 gms. of sugar per day in the urine, his blood sugar was 0·3 per cent, and acetone and diacetic acid were abundant. He gradually improved as the sepsis diminished, the urinary sugar and blood sugar falling considerably, but on 18th December, 1922, there was a recurrence of sepsis in the palm, and the urinary sugar went up to 550 gms. and the blood sugar to 0·29 per cent. The hand was incised and rapidly healed, the urinary sugar and blood sugar again fell, and he was dismissed on 17th January, 1923, with the hand well healed, and with a daily output of only 20 gms. of sugar.

On 21st March, 1923, he was readmitted for insulin treatment, and dismissed sugar free on 35 U.



On 27th June, 1923, he was again readmitted, this time with a discharge from the ear. The urinary sugar was 250 gms. in the day, insulin was given, and he became sugar free on 30 U.

2. The second case, after treatment with insulin, was dismissed sugar free in June, 1923, without insulin. He was well till readmitted on 3rd October, 1923, with a carbuncle on the neck; the urinary sugar was then 220 gms., the blood sugar 0.35 per cent. Insulin brought the sugar down to *nil*, but a dose of 35 U was required, showing that his tolerance was greatly reduced since his last admission.

After studying cases of this kind it is only natural to ask—Whether or not sepsis in a normal individual leads to any such want of carbohydrate tolerance?

It has been observed by a number of workers on this subject that in certain abnormal conditions, other than diabetes, there is a distinct defect in carbohydrate tolerance, evidenced either by the actual presence of glycosuria or by the appearance of sugar in the urine following upon the ingestion of a quantity of glucose. In the *Lancet* of 23rd December, 1899, Cuthbert Wallace described a series of cases of this kind—some were cases of gangrene, others were septic conditions, such as carbuncle, cellulitis, &c. In all of these he found either actual glycosuria or potential glycosuria, and he pointed out that as soon as the septic focus was removed, *e.g.*, on amputation of a gangrenous foot, the glycosuria disappeared. Prévost, in 1878, and Rédaud, in 1886, had previously described similar cases, calling the condition of "sapræmic glycosuria."

Since then, methods of estimating the percentage of sugar in the blood have made it possible to observe more accurately what actually occurs in cases such as the above. Grigaud, Brodin, and Rouzaud, in 1920, found that hyperglycæmia was the rule in infectious diseases, while Poll and Campagnolle showed that there was a lowered glucose tolerance in such cases. Cammidge finds that even in an ordinary febrile cold the fasting level of the blood sugar is raised, and glucose tolerance is lowered.

Willcox and others have drawn attention to the influence of septic foci in causing hyperglycæmia. Willcox draws special attention to the frequency with which glycosuria is associated

**SYNOPSIS OF CASES**

**INSULIN TREATMENT**

**"NEW CASES"**

**MALE**

**I. 1 - I. 37**

**FEMALE**

**I. 38 - I. 67.**

Case	Age	Diabetic	Lag	Renal	Diabetic without Symptoms	Not Diabetic	Emaciated	Acid	Weight	Immed. Insulin Max. Dis.	Later Insulin Max. Dis.	No Insulin	Remarks
I. 1	31	+	-	-	-	-	-	+	+3	85 30	-	-	
I. 2	53	+	-	-	-	-	-	+	+3	60 45	-	-	
I. 3	41	+	-	-	-	-	-	+	-1	-	-	+	
I. 4	57	-	-	-	+	-	-	-	-1.5	-	-	+	Tabes Dorsalis.
I. 5	63	+	-	-	-	-	+	-	0	25 20	-	-	
I. 6	65	+	-	-	-	-	-	+	0	40 25	-	-	
I. 7	50	+	-	-	-	-	-	+	+1	20 20	-	-	
I. 8	25	+	-	-	-	-	-	+	0	-	-	+	Dismissed Irreg.
I. 9	14	+	-	-	-	-	-	+	-2	-	-	+	
I. 10	23	+	-	-	-	-	-	+	-2	-	40 40	-	
I. 11	29	+	-	-	-	-	-	-	-3	-	20 20	-	
I. 12	20	+	-	-	-	-	-	+	-4	20 0	-	-	
I. 13	35	+	-	-	-	-	-	+	-2	20 0	-	-	
I. 14	19	+	-	-	-	-	-	+	0	45	-	-	Death.
I. 15	8	-	+	-	-	-	-	-	0	-	-	+	
I. 16	42	+	-	-	-	-	-	+	-1	-	30 30	-	
I. 17	40	+	-	-	-	-	-	+	-2	-	50 50	-	
I. 18	45	+	-	-	-	-	-	+	0	25 3	-	-	
I. 19	15	+	-	-	-	-	-	+	3	40 10	-	-	
I. 20	32	+	-	-	-	-	-	-	-1	-	35 20	-	
I. 21	53	+	-	-	-	-	-	+	-2.5	-	15 10	-	
I. 22	60	-	-	-	+	-	-	-	0	-	10 5	-	Syphilis.
I. 23	64	+	-	-	-	-	-	-	-3	-	-	+	

Case	Age	Diabetic	Lag	Renal	Diabetic without Symptoms	Not Diabetic	Emaciated	Acid	Weight	Immed. Insulin Max. Dis.	Later Insulin Max. Dis.	No Insulin	Remarks
I. 24	31	+	-	-	-	-	-	-	0	-	10 0	-	
I. 25	53	-	-	-	+	-	-	-	-2	-	-	+	Ch. Inter. Nephrit.
I. 26	53	+	-	-	-	-	+	-	+1	30 30	-	-	Phthisis.
I. 27	53	+	-	-	-	-	-	-	+1	-	25 25	-	
I. 28	60	+	-	-	-	-	+	-	+2.5	35 35	-	-	
I. 29	49	-	-	+	-	-	-	-	0	-	-	+	
I. 30	48	+	-	-	-	-	-	-	+0.5	-	10 10	-	
I. 31	64	+	-	-	-	-	-	-	+1	-	-	+	
I. 32	36	-	-	-	+	-	-	-	0	-	-	+	
I. 33	30	-	+	-	-	-	-	-	0	-	10 0	-	
I. 34	54	+	-	-	-	-	-	-	-0.5	-	-	+	
I. 35	47	+	-	-	-	-	-	-	-1	-	25 25	-	
I. 36	68	+	-	-	-	-	-	-	0	45 45	-	-	
I. 37	51	+	-	-	-	-	-	+	+1	55 55	-	-	

Case	Age	Diabetic	Lag	Renal	Diabetic without Symptoms	Not Diabetic	Emaciated	Acid	Weight	Immed. Insulin Max. Dis.	Later Insulin Max. Dis.	No Insulin	Remarks
I. 38	47	+	-	-	-	-	-	-	-5	-	-	+	Broncho.Pneu.
I. 39	71	-	-	-	+	-	-	+	-2	-	-	+	Senility.
I. 40	28	-	-	+	-	-	-	-	0	-	-	+	
I. 41	46	+	-	-	-	-	+	+	0	(2) 60	(1) 15 15	-	Two Ads.:Death.
I. 42	66	-	-	-	+	-	-	-	0	-	-	+	Cystitis.
I. 43	24	+	-	-	-	-	-	+	0	-	50 45	-	
I. 44	42	+	-	-	-	-	+	-	0	-	25 10	-	
I. 45	22	+	-	-	-	-	-	+	-1	-	10 0	-	
I. 46	25	-	-	+	-	-	-	-	0	-	-	+	
I. 47	55	+	-	-	-	-	-	-	-2	-	-	+	
I. 48	64	-	-	-	+	-	-	-	-2	-	-	+	Cystitis:Rheumat.
I. 49	27	+	-	-	-	-	-	-	+3.5	-	35 35	-	
I. 50	59	+	-	-	-	-	-	-	0	-	35 35	-	Sepsis.
I. 51	58	+	-	-	-	-	-	-	-5	-	-	+	
I. 52	15	+	-	-	-	-	-	-	+1.5	-	-	+	
I. 53	37	+	-	-	-	-	-	-	+3	-	35 20	-	Cardiac-mitral.
I. 54	56	+	-	-	-	-	-	-	-5	-	5 5	-	
I. 55	42	-	-	-	+	-	-	-	+1	-	-	+	
I. 56	9	+	-	-	-	-	+	-	+1	-	-	+	
I. 57	31	+	-	-	-	-	-	+	-2.5	-	30 15	-	
I. 58	52	+	-	-	-	-	-	-	-3	-	-	+	
I. 59	58	+	-	-	-	-	-	-	-1.5	-	-	+	
I. 60	58	+	-	-	-	-	-	+	-1	50 0	-	-	

Case	Age	Diabetic	Lag	Renal	Diabetic without Symptoms	Not Diabetic	Emaciated	Acid	Weight	Immed. Insulin Max. Dis.	Later Insulin Max. Dis.	No Insulin	Remarks
I. 61	53	+	-	-	-	-	-	-	+1	-	-	+	
I. 62	51	+	-	-	-	-	-	+	+1	-	25 25	-	
I. 63	42	+	-	-	-	-	-	-	-1.5	-	-	-	
I. 64	57	+	-	-	-	-	-	-	+1.5	-	15 10	-	
I. 65	41	+	-	-	-	-	-	-	-2	-	25 25	-	
I. 66	61	+	-	-	-	-	-	-	-1	-	-	+	
I. 67	62	+	-	-	-	-	-	+	+2	40 40	-	-	

R. M.

Age 31.

Commercial Traveller.

ADMITTED: 21:4:23.

PRESENT ILLNESS: Duration: 4 years.

Symptoms: Thirst, polyuria, loss of weight.

Sugar discovered Oct. 1920: dieted with poor results.

PAST ILLNESSES: Appendix removed 1913.

Malaria ~~1917~~ 1918.

FAMILY HISTORY: Negative.

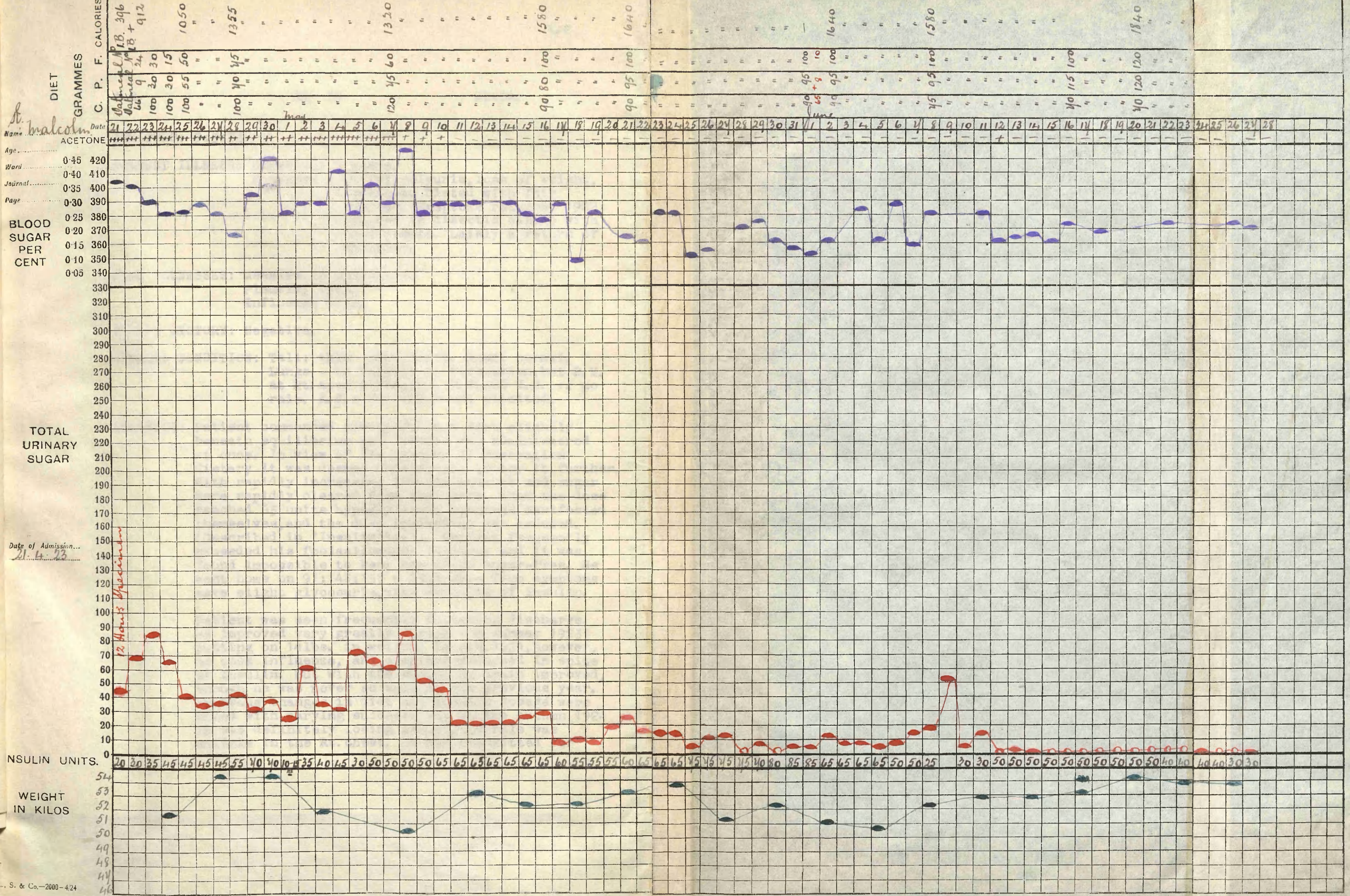
GENERAL CONDITION: Tall: spare. Skin dry and yellow tinged. Heart normal: pulse frequent and weak. Lungs normal: odour of acetone in breath: some "air-hunger." Teeth septic: tongue furred. K.J.s not elicited: pupils normal.

PROGRESS: Patient was so collapsed on admission and shewed such evidence of acidosis that insulin was commenced at once with oatmeal diet. Cardiac stimulants (digitalis, alcohol, etc.) were also administered. The general condition rapidly improved, the pulse falling to normal in 4 days and the tongue cleaning. Acetone, however, persisted in amount in the urine, but the alveolar CO<sub>2</sub> was more satisfactory. There was definite oedema on the 27th. Insulin was increased to 70 units per day, and continued thereafter in rather smaller amount: but the urine was not rendered acetone-free for 19 days, the longest period which I have found it to persist. On 9:5:23 the K.J.s were elicited. Despite changes in the diet and insulin dosage sugar persisted in the urine and the B.S. ran above the normal level. A maximum dose of 85 units was reached. At this point the patient had a definite hypoglycaemic reaction, abolished by sweet tea and bread. Following this the dose of insulin was reduced, and with continued dosage of 50 units the urine was rendered sugar-free. He left hospital on 70:120:120 = 1840 with 30 units of insulin, free from symptoms.

	Admission	DISCHARGE
Weight	50.8k.	53.7k.
Blood Sugar	0.38%	0.21%
Urinary Sugar	70gms.	Trace
" Acetone	++++	-

A satisfactory result in a difficult case. When seen on 12:1:25 he was very well in every way.







A.R.

Age 53

Clerk.

ADMITTED: 9:5:23.

PRESENT ILLNESS: Duration: 2 years.

Symptoms : Thirst, polyuria, loss of weight, Weakness. Dieted with fair success. Symptoms returned 1922: treated in diabetic clinic with good result. Lately a return of symptoms.

PAST ILLNESSES: Measles in childhood.  
Pleurisy 1906.  
Influenza 1918.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Tall: thin. Skin moist. Heart normal.  
Lungs - no dulness to percussion, but R.M. at Rt. apex deficient, V.F. and V.R. +: no rale. K.J.s and A.J.s not elicited.

PROGRESS: Patient commenced treatment on a diet slightly beneath equilibrium and insulin was administered at once. In view of the history of starvation dietary it was deemed unnecessary to try it further. With rapidly increasing insulin acetone and sugar were rapidly cleared from the urine. When the dose reached 65 units hypoglycaemic symptoms manifested themselves, and the dose thereafter was reduced. (Described in 'Insulin' text). Patient frequently exceeded his food allowance latterly and it was found impossible to keep the urine sugar-free. He went home on 91:143:155 = 2351, free from symptoms save slight glycosuria, with 45 units of insulin.

Patient was seen frequently following discharge. He improved very greatly during the summer 1923, putting on 14 lbs. in weight. In Jan. 1924, however, he took Influenza, and symptoms returned in spite of insulin. But with the summer he again improved, though he was never so well as the previous year. Numerous changes in diet and insulin dosage were tried with varying success: but in the autumn 1924 he was definitely losing ground, and rale was audible in the Rt. chest. He was readmitted for

PROGRESS: treatment.

(Contd.)

READMITTED: 6:11:24. The general condition was little changed from 18 months earlier. The K.J.s were now present and fairly active. Considerable rale at Rt.apex.

PROGRESS: Diets were carefully revised, and on this occasion patient ate nothing beyond his rations. All symptoms rapidly disappeared, and patient went home on 57:51:151 = 1971, free from symptoms with 40 units of insulin. By the end of Dec. 1924 he had gained 12lbs. The rale at the Rt.apex was no longer audible.

	1923		1924	
	Ad.	Dis.	Ad.	Dis.
Weight	59k.	63k.	63.7k.	69k.
Blood Sugar	9.28%	0.15%	-	0.13%
Urinary Sugar	245gms.	20gms.	2%	Free
Urinary Acetone	++	-	-	-

An excellent result in a case of severe diabetes.



# DIET

GRAMMES

Date \_\_\_\_\_

ACETONE

0.45 420

0.40 410

0.35 400

0.30 390

0.25 380

0.20 370

0.15 360

0-10 330  
0-11 310

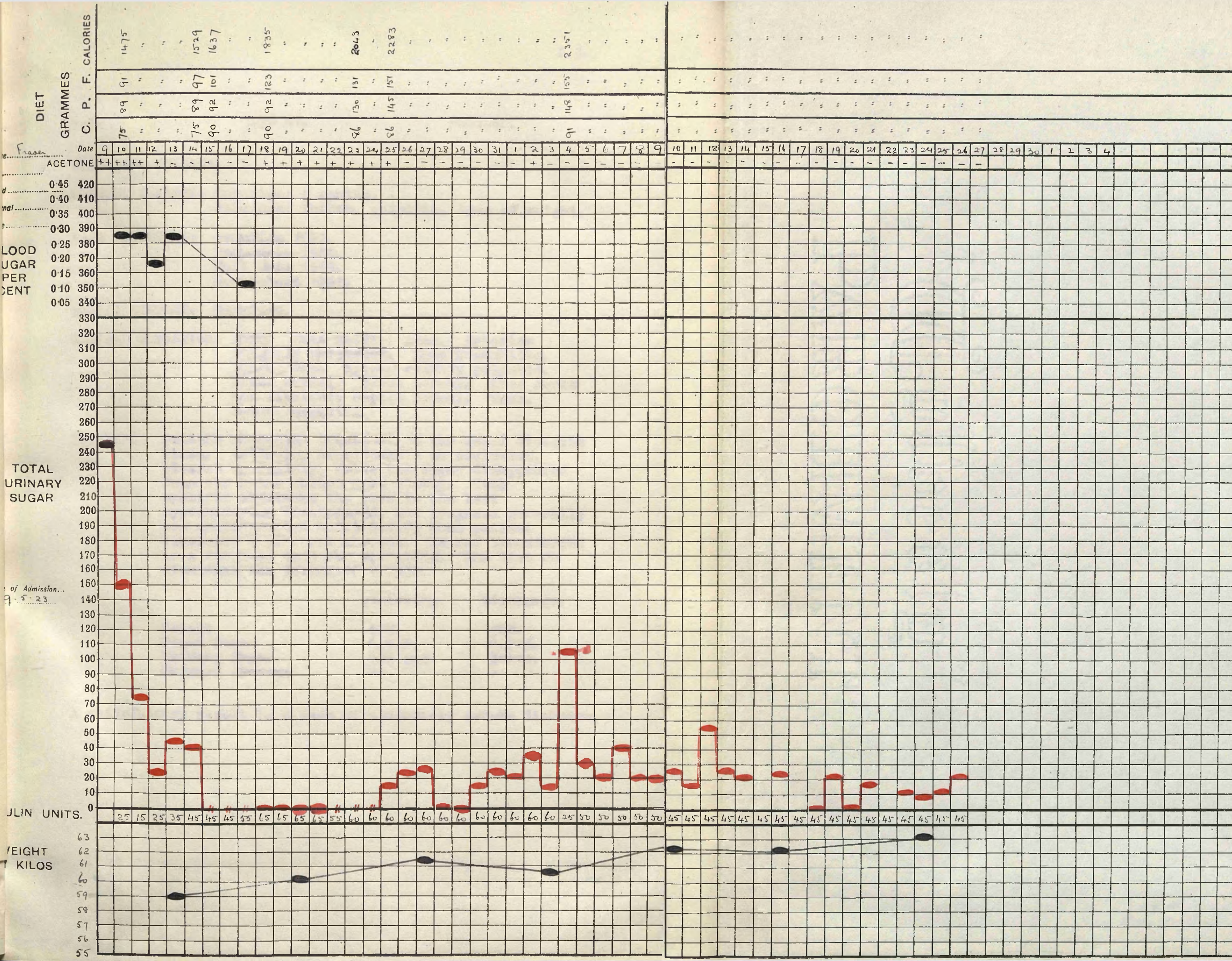
003 070

TOTAL  
URINARY  
SUGAR

of Admission..  
9.5.23

PLAN UNITS.

EIGHT  
KILOS





G.C.

Age 41.

Puddler.

ADMITTED: 27:6:23.

**PRESENT ILLNESS:** Duration: 3 months.  
Symptoms: Thirst, polyuria, loss of weight.

**PAST ILLNESSES:** Jaundice 1887.  
Pneumonia 1900.  
Soft Sore 1918.  
Gonorrhoea 1921.

**FAMILY HISTORY:** Negative.

**GENERAL CONDITION:** Thin. Skin moist. Acne. Arteries slightly thickened. Some Bronchitis. Tongue dry. Spleen readily palpable. Liver normal. Blood normal. Knee Jerks not elicited: Pupils normal. Wass. React. negative.

**PROGRESS:** Patient commenced treatment on the usual dietetic lines. Acidosis, considerable on admission, cleared up rapidly, while the sugar disappeared from the urine though more slowly. With specific treatment the mass in the left hypochondrium disappeared, and progress generally was uninterrupted until 2:8:23 when patient developed a perineal abscess. He was transferred to a surgical ward for operation. The diet on dismissal was 80:80:140 = 1900.

	Admission.	Discharge.
Weight	51k.	50k.
Blood Sugar	0.47%	0.21%.
Urinary Sugar	224 gms.	Trace.
Urinary Acetone	++	+

A satisfactory result in a case of moderately severe diabetes.







D.D.

Age 57

Clerk.

ADMITTED: 7:7:23.

PRESENT ILLNESS: Duration: Some years.

Symptoms: Weakness of left leg and arm, and of right leg.

Sugar discovered on routine examination in hospital.

PAST ILLNESSES: Syphilis 1898.

Malaria 1916.

FAMILY HISTORY: Mother and one brother died of Phthisis.

GENERAL CONDITION: Stout. Skin dry. Psoriasis. Heart normal. B.P. 150 mm.Hg. Lungs normal. Tongue coated: pyorrhoea. K.J.s absent: plantar reflexes extensor: Argyll-robertson pupils. Rhomberts sign +. Walks with a limp, dragging the leg. Incoordination hands and feet. Mentally very dull. Wass.React. + ve.

PROGRESS: Patient was never strictly dieted and glycosuria persisted throughout his whole residence. On light diet with sugar the urinary sugar rose to 240 gms. When sugar was withdrawn from this diet it fell to 30 gms. The morning blood sugar reading was never found above 0.10%: but a Glucose Test on 13:7:23 gave a 'Diabetic' curve. Patient left hospital on 117:78:46 = 1194, his condition practically unchanged.

	Admission	Discharge
Weight	59.5k.	58.1k.
Blood Sugar	0.15%	0.27%
Urinary Sugar	95gms.	8gms.
Urinary Acetone	-	-

A case of Tabes Dorsalis with a mild diabetes.







W.M.B.

Age 63

Grocer.

ADMITTED: 25:7:23.

PRESENT ILLNESS: Duration: 8 years.

Symptoms: Weakness, sleeplessness, anorexia.  
Sugar discovered 8 years ago: no treatment.

PAST ILLNESSES: Nil save results of alcoholism.

FAMILY HISTORY: Negative.

PRESENT CONDITION: A frail, done old man. Poorly nourished. Ill and worried. Heart enlarged: V.S. murmur at apex, conducted. Arteries sclerosed: B.P. 225 mm. Hg. Tongue furred. Reflexes ++. Urine-albumen ++.  
Unable to walk on admission.

PROGRESS: Glycosuria was considerable on admission, and shewed little change on a moderately restricted diet - ( 78:74:86 ). Insulin was commenced on the 5th. day, and increased with increasing diet. Thereafter progress was very satisfactory. The tongue cleaned, appetite returned, and patient slept well. The B.P. fell to 180 mm. Hg. He felt better than he had done for years, and latterly was able to be about for the greater part of the day. He left the Home on 132:89:139 = 2099, free from discomfort. On 20 units of insulin the urinary sugar ran under 20 gms. in 24 hrs. No attempt was ever made at starvation on account of age and weakness.

A satisfactory result in an old diabetic. The glycosuria was efficiently controlled by insulin, and symptoms held in check. Patient carried on very comfortably for two years when he died of pneumonia.



**CALORIES**

Date \_\_\_\_\_

ACETONE

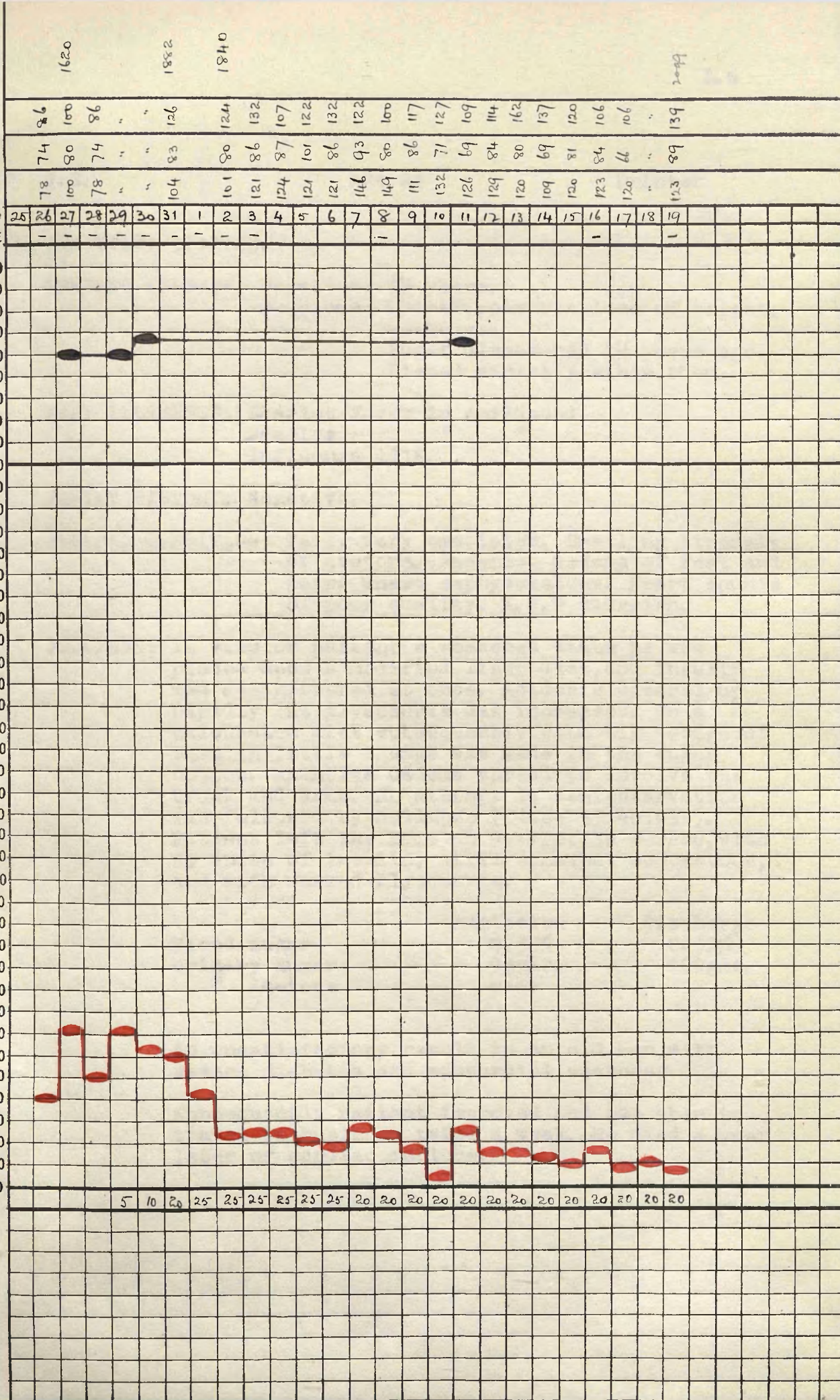
LOOD  
UGAR  
PER  
CENT

TOTAL  
URINARY  
SUGAR

of Admission...

LIN UNITS.

EIGHT  
KILOS



J.R.

Age 65

Printer

ADMITTED: 31:7:23.

PRESENT ILLNESS: Duration: 2½ years.

Symptoms: Thirst, polyuria, loss of weight, weakness.

Sugar discovered 2½ years ago.

Dieted strictly since then.

PAST ILLNESSES: Scarlet Fever in childhood.

Measles " "

Influenza 1918.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Tall: very emaciated. Smelling strongly of acetone. Anaemic. Oedema of feet and legs. Chest emphysematous. Heart sounds of poor quality. K.J.s sluggish.

PROGRESS: In view of patient's weakened state he was placed upon a modified light diet, and insulin was administered at once. Acidosis cleared up rapidly but glycosuria was increased. On a calculated diet subsequently with big dosage of insulin little change was made in the sugar output. Meantime oedema spread to involve the trunk and arms. An attempt at semi-starvation was followed by collapse ( diet 33:40:45 ). Patient left the Home on 99:120:156 = 2280, with 25 units of insulin, still somewhat oedematous, and with marked glycosuria.

	Admission	Discharge
Blood Sugar	0.35%	0.37%
Urinary Sugar	95gms.	120gms.
" Acetone	++++	-

An unsatisfactory result in an old man with severe diabetes and myocardial weakness.

Subsequently patient improved, and was able to travel to business twice a week. He died a year later of cardiac failure.





J.B.P.

Age 50

Motor Driver.

Treated at home.

PRESENT ILLNESS; Duration: 4 years.

Symptoms: Thirst, polyuria, weakness. Dieted with some improvement. Recently dimness of vision-toxic amblyopia. Cannot read or write. Loss of weight.

Past Illnesses: Nil.

FAMILY HISTORY: An uncle has diabetes.

GENERAL CONDITION: Fairly well nourished. Skin moist. Reflexes normal. Physical examination entirely negative.

PROGRESS: Patient was placed upon an equilibrium diet and insulin was commenced at once. Acetone quickly disappeared from the urine, and glycosuria was easily controlled. He was very careless about diet and occasionally exceeded his allowance. He completed treatment on 51:117:110 = 1662, free from symptoms, with 20 units of insulin.

	Admission	Discharge
Weight	57.2k.	58.2k.
Blood Sugar	0.30%	0.17%
Urinary Sugar	10gms.	Free
" Acetone	++	-

Patient was seen frequently during the following 6 months. His weight increased to 61.8k. and his eyesight improved so that he was able to write and read with ease.

A satisfactory result in a careless patient with moderately severe diabetes.



# DIET

## GRAMMES

C. P. F. CALORIES

74	74	101	1501
70	123	107	1735
57	102	65	1221
55	175	155	2315
57	117	110	1662

Date 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16

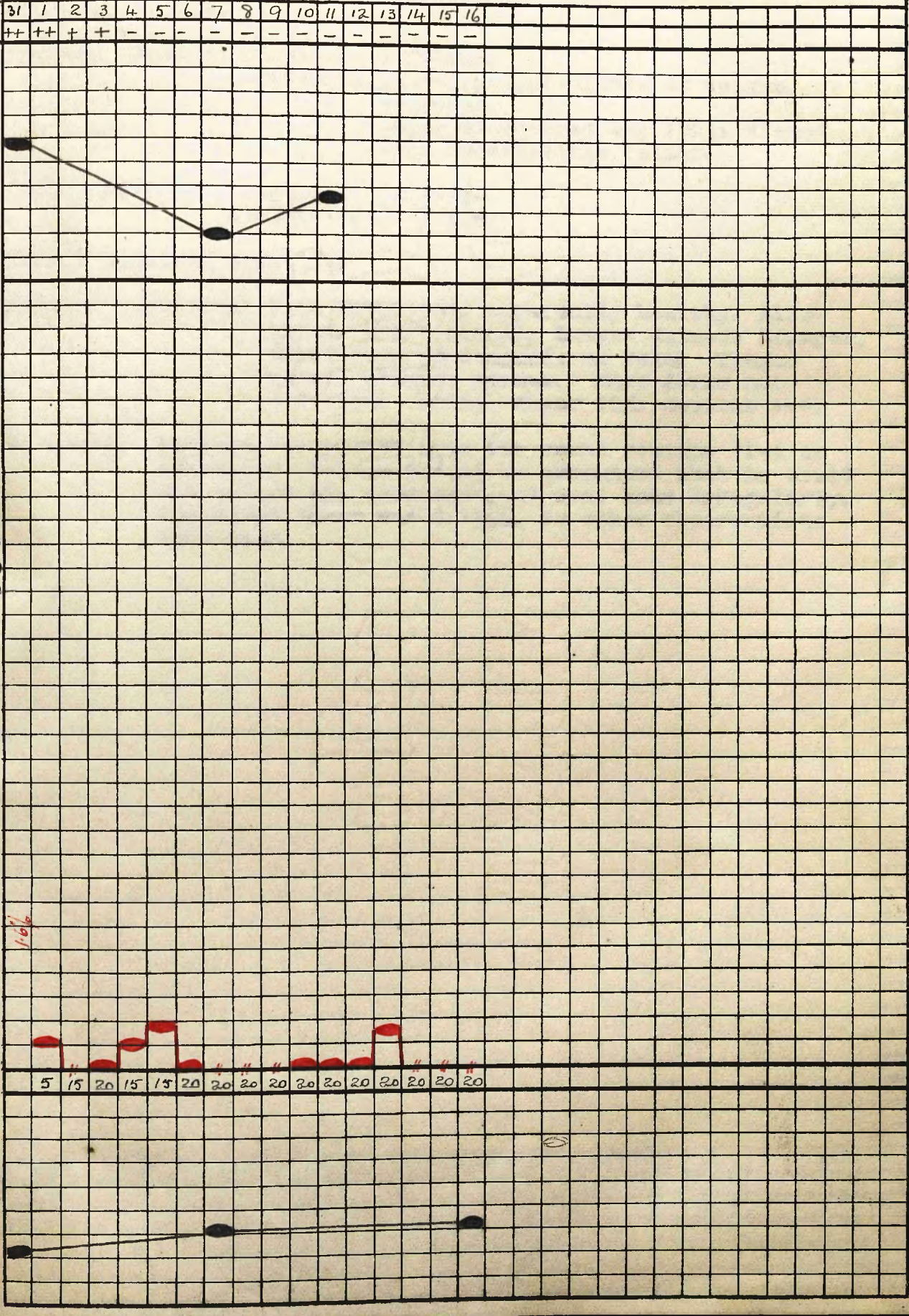
ACETONE  
 0.45 420  
 0.40 410  
 0.35 400  
 0.30 390  
 0.25 380  
 0.20 370  
 0.15 360  
 0.10 350  
 0.05 340

## TOTAL URINARY SUGAR

Time of Admission... 11:7:23

## ULIN UNITS.

## WEIGHT KILOS



J.W.

Age 25

Grocer.

**ADMITTED:** 7:8:23.**PRESENT ILLNESS:** Duration: 9 months.

Symptoms: Thirst, polyuria, loss of weight, weakness.

Sugar discovered May 1923: dieted with considerable relief.

**PAST ILLNESSES:** Measles in infancy.

Gassed 1915: ? T.B.

**FAMILY HISTORY:** Negative.**GENERAL CONDITION:** Well developed: apparently healthy. Skin moist. Heart normal. Lungs- dulness Rt. apex, expiration prolonged: no rale. Tongue moist: abdomen normal. Knee Jerks not elicited. Urine- sugar 10%: acetone +++.**PROGRESS:** Patient was placed upon the usual routine diet on admission: but on 9:8:23 he announced that he could not endure the treatment, and went home irregularly. The Blood Sugar was 0.156%. No other observations were made.



DIET		GRAMMES		C. P. F. CALORIES	
Name <u>J. Walker</u>		Date <u>7 8 9</u>		385 "	
Age <u>25</u>		ACETONE <u>+++++</u>		5 "	
Ward		0.45	420	25 "	
Journal		0.40	410		
Page		0.35	400		
		0.30	390		
BLOOD		0.25	380		
SUGAR		0.20	370		
PER		0.15	360		
CENT		0.10	350		
		0.05	340		
			330		
			320		
			310		
			300		
			290		
			280		
			270		
			260		
			250		
			240		
TOTAL			230		
URINARY			220		
SUGAR			210		
			200		
			190		
			180		
			170		
			160		
Date of Admission...			150		
<u>7-8-13</u>			140		
			130		
			120		
			110		
			100		
			90		
			80		
			70		
			60		
			50		
			40		
			30		
			20		
			10		
			0		
INSULIN UNITS.					
WEIGHT		60			
IN KILOS		59			
		58			
		57			
		56			
		55			

R.T.

Age 14

Leather-worker.

ADMITTED: 9:8:23.

PRESENT ILLNESS: Duration: 6 weeks.

Symptoms: Thirst, polyuria, loss of weight, weakness.

Sugar discovered July 1923:

dieted without relief of symptoms.

Past Illnesses: Whooping Cough in childhood.  
Chicken Pox 1913 .

FAMILY HISTORY: Negative.

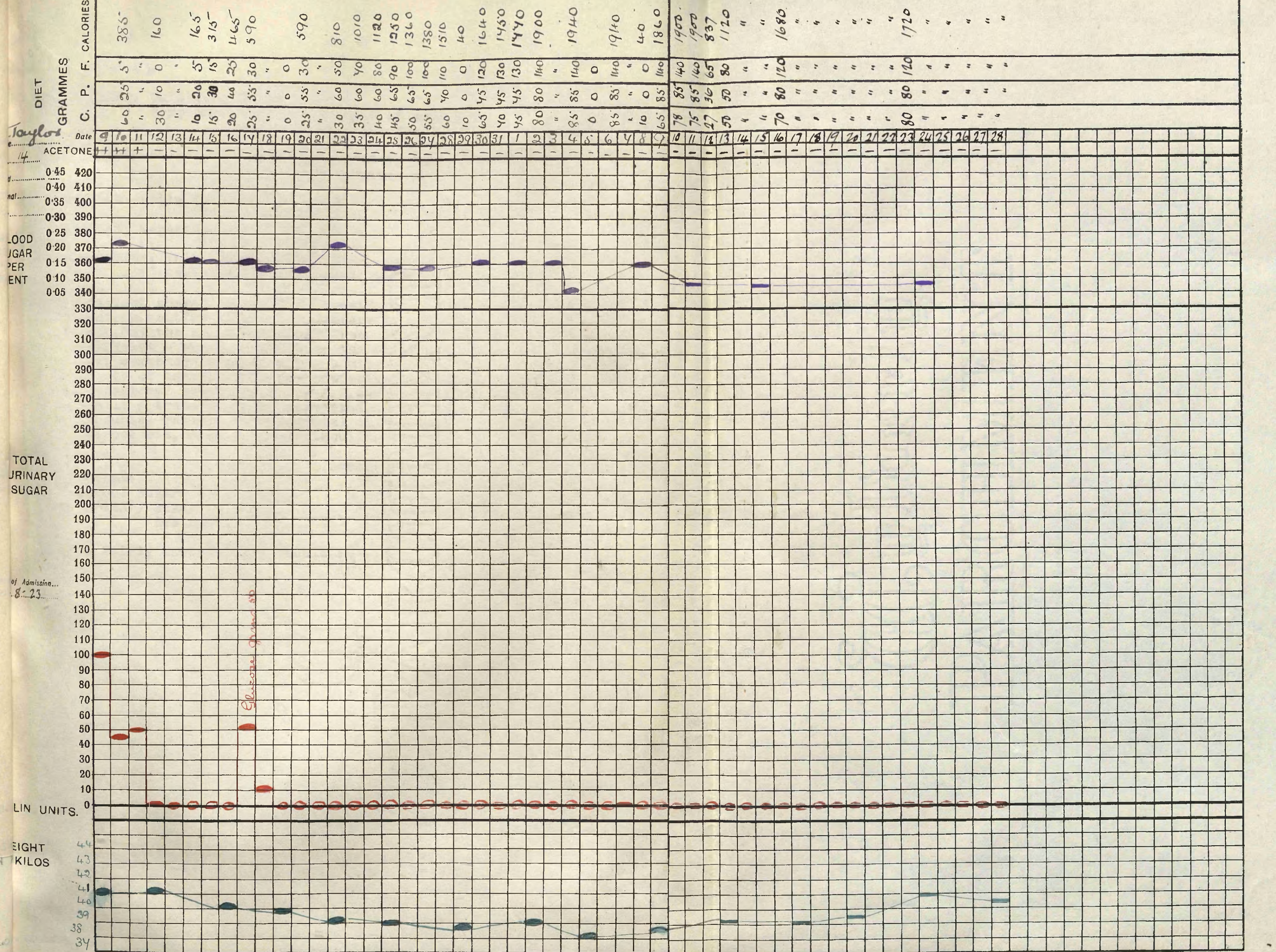
GENERAL CONDITION: Well developed. Skin dry but clear.  
Heart normal. Lungs healthy. Tongue dry:  
Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic lines, and acetone and sugar rapidly disappeared from the urine. A Glucose Test on 17:8:23 gave a 'Diabetic curve'. Progress was uninterrupted, and patient left hospital on 80:80:120 = 1720, free from symptoms.

	Admission	Discharge
Weight	41.2k.	39.6k.
Blood Sugar	0.14%	0.07%
Urinary Sugar	98gms.	Free
"    Acetone	++	-

A very satisfactory result in a case of moderate severity.







David Glassford

Age 23

Draper.

ADMITTED: 17:8:23.

PRESENT ILLNESS: Duration:  $3\frac{1}{2}$  years.

Symptoms: Progressive loss of energy, thirst, frequency of micturition, loss of weight, feeling of numbness in legs.

Sugar discovered in 1920: treated successfully with diet. Symptoms returned 3 weeks ago.

PAST ILLNESSES: Boils 1917, in the Army.

FAMILY HISTORY: Negative.

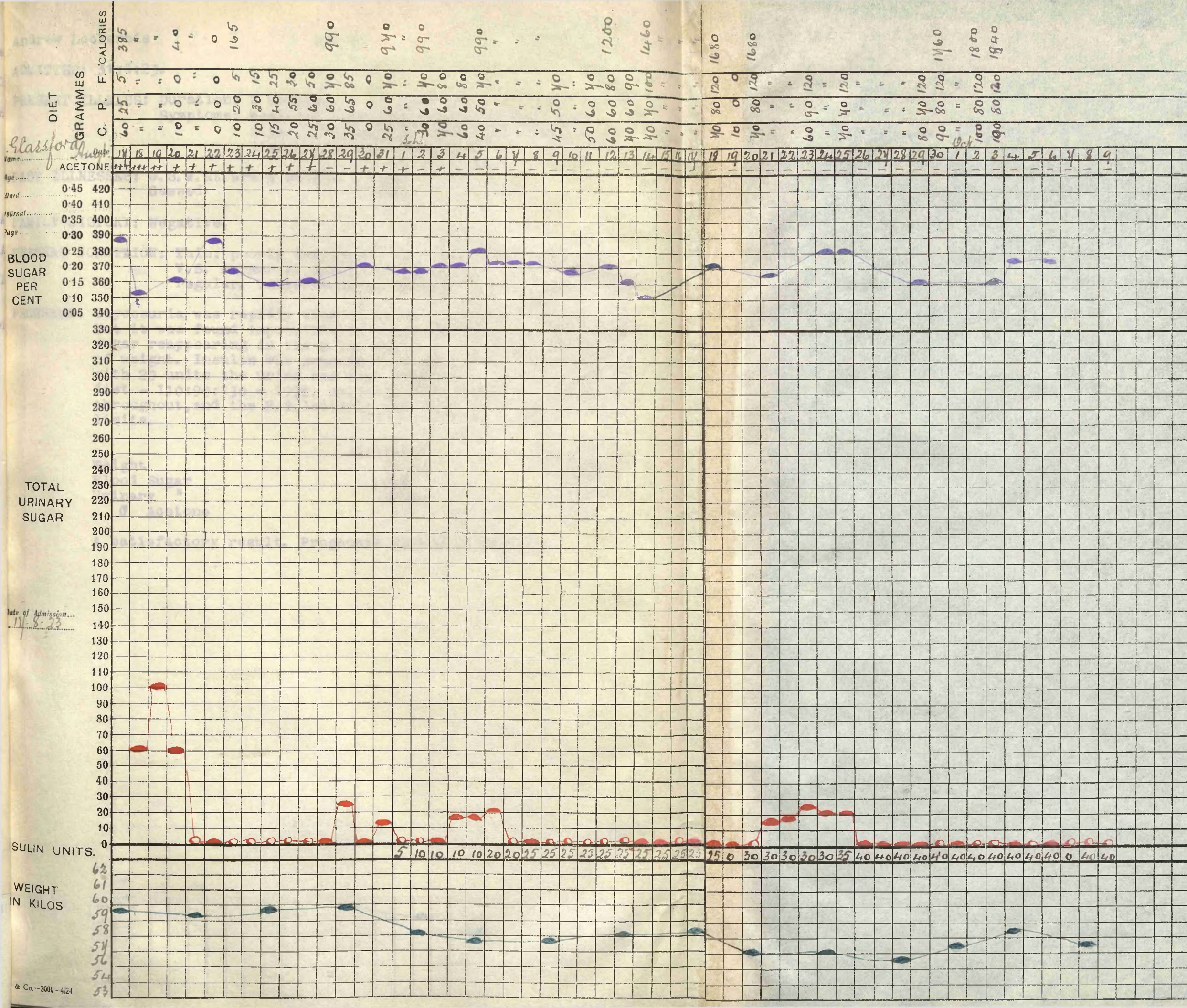
PRESENT CONDITION: Thin: in poor condition. Skin moist: no skin lesions.  
Heart and lungs normal. Reflexes normal. Eyes normal.

PROGRESS: Patient shewed evidence of considerable acidosis on admission, but on routine starvation dietary it rapidly cleared up. By the 4th. day the urine was sugar-free. As carbohydrate tolerance was very low - sugar appeared in the urine on 25-30gms.C. - insulin was commenced 2 weeks after admission. In spite of increasing doses of insulin progress thereafter was very slow. Even with occasional starve days the B.S. tended to run above normal limits, while a trace of sugar persisted in the urine. Acidosis caused no trouble. Patient left hospital on 100:80:120 = 1800. free from glycosuria, on 40 units of insulin.

	Admission	Discharge
Weight	59.6k.	57.6k.
Blood Sugar	0.258%	0.22%
Urinary Sugar	60gms.	Free.
" Acetone	+++	-

A fairly satisfactory result in a severe case.







Andrew Lookerbie

Age 29

Grocer.

ADMITTED: 31:8:23.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Weakness, loss of weight, thirst, polyuria.

Sugar discovered 2 weeks ago: dieted with disappearance of symptoms.

PAST ILLNESSES: G. S. W. Rt. arm ( slight ) 1916.

Gassed

1917.

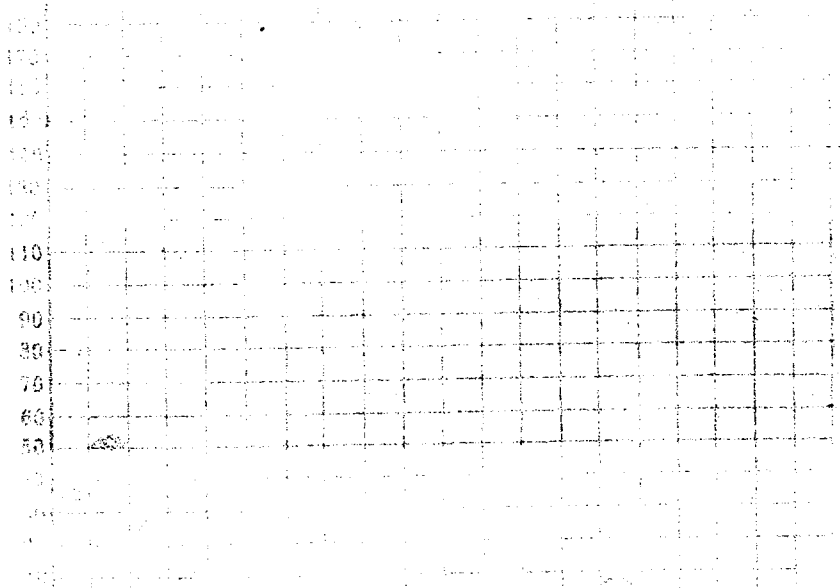
FAMILY HISTORY: Negative.

PRESENT CONDITION: Thin: poorly developed. Skin dry. Heart not enlarged:  
V. S. murmur at apex, propagated into axilla: pulse  
regular. Lungs healthy. Abdomen normal. Reflexes normal.

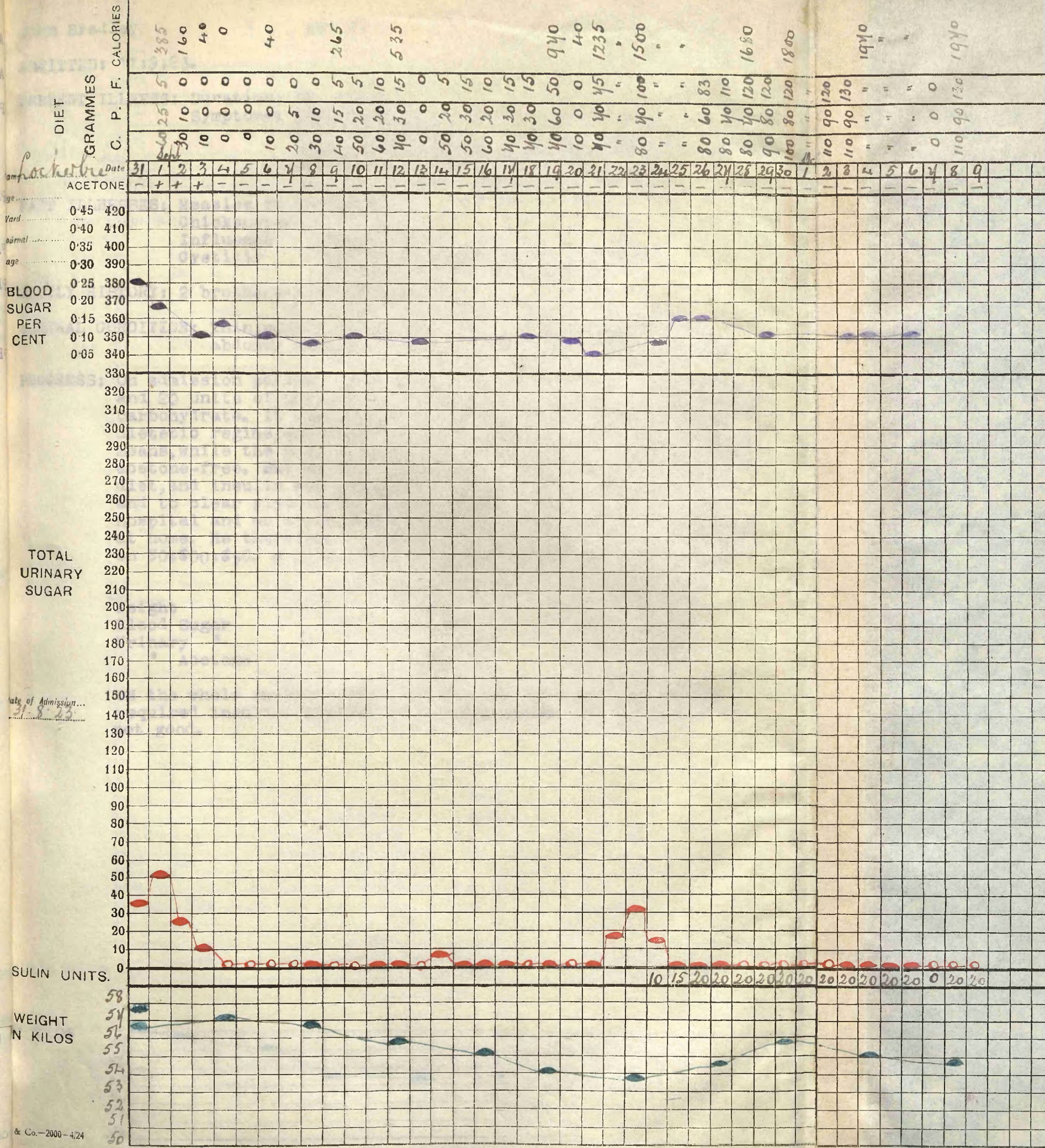
PROGRESS: Glycosuria was rapidly cleared up on the usual dietetic lines, but it was found impossible to reach an adequate diet without sugar reappearing in the urine and there was considerable loss of weight. Insulin was commenced 24 days after admission, and with 20 units the urine was kept sugar-free on a satisfactory diet - 110:90:130 = 1970. Acidosis gave rise to no trouble throughout, and the B. S. latterly was constantly within normal limits.

	Admission	Discharge
Weight	56.8k.	54½k.
Blood Sugar	.25%	.110%
Urinary "	50gms.	Free.
Acetone	-	-

A satisfactory result. Prognosis good with insulin.









John Bradley

Age 20

Pithead-worker.

ADMITTED: 11:9:23.

PRESENT ILLNESS: Duration: 18 months.

Symptoms: Thirst, frequency of micturition, weakness, loss of weight.

Sugar discovered 6 months ago. Dieted with fair success, but weight still falling.

PAST ILLNESSES: Measles in childhood.

Chicken-pox "

Influenza 1921.

Cystitis 1923. ( 2 months before admission )

FAMILY HISTORY: 2 brothers died of T.B.

GENERAL CONDITION: Thin: poorly developed. Skin moist. Heart, lungs, and abdomen shewed no abnormality. Knee jerks present.

PROGRESS: On admission patient shewed evidence of considerable acidosis, and 20 units of insulin were administered at once with abundant carbohydrate. It was then considered safe to revert to ordinary dietetic regime, and glycosuria was readily cleared by this means, while the B.S. fell to normal limits, and the urine became acetone-free. But weight fell steadily in spite of increasing diet, and insulin was resumed to allow of more rapid progress and to clear glycosuria. Unfortunately patient had to leave hospital and no arrangements could be made to continue insulin at home. He therefore reverted to diet alone and was dismissed on \$0:\$00:\$50. = 2070 , free from glycosuria but losing weight.

	Admission	Discharge
Weight	58k.	54k.
Blood Sugar	0.40%	0.18%
Urinary "	140gms.	Free.
" Acetone	+++	-

ON the whole an unsatisfactory result. This patient really required insulin. Without it ~~was not good~~ the prognosis was not good.







J. L.

Age 35.

Clerk.

ADMITTED: 25:9:23.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, weakness.

Carbuncle on right cheek opened two days before admission. Sugar discovered 1921. Dieted with improvement.

PAST ILLNESSES: Nil.

FAMILY HISTORY: Good,

GENERAL CONDITION: Spare build. Skin healthy save for small carbuncle on right lower cheek with recent crucial incision. Nothing else abnormal discovered on physical examination.

PROGRESS: In view of the presence of the carbuncle and considerable acidosis insulin was administered at once. Wet boracic dressings were applied to the cheek. After three days on insulin the acidosis completely disappeared and the slough quickly separated. The inflammatory condition subsided with remarkable rapidity. Thereafter treatment proceeded on the usual dietetic lines. Patient left the home on 90:85:135  $\Delta$  1915 without insulin, with a normal fasting blood sugar, and free from symptoms. The carbuncle was completely healed.

	Admission.	Discharge.
Weight	64k	62k.
Blood Sugar	0.25%	0.12%.
Urinary Sugar	50 gms.	Free.
Urinary Acetone	++.	-

Patient was seen on 14:4:24 when he was very well.  
His weight was 70k.





D. F.

Age 19

Clerk.

ADMITTED: 28:9:23.

PRESENT ILLNESS: Duration 2 years.

Symptoms : Thirst, polyuria, weakness, loss of weight.

Coma averted with difficulty 2 years before. Well till 3 weeks ago. Sudden collapse before admission.

PAST ILLNESSES: Nil save childhood ailments.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Very emaciated. Skin moist. Collapsed and cold. Pulse frequent, small, and soft. Respirations 32 per min., full and sighing in character: odour of acetone in breath. Nothing abnormal detected in heart or lungs. Urine scanty: albumen +++: sugar 5%: acetone +++: diacetic acid +.

PROGRESS: Patient was rather drowsy but answered questions intelligently. Under stimulation and insulin he made some improvement and became warmer while the pulse improved in quality: but toward evening he vomited and the pulse began to fail. Meantime the drowsiness practically disappeared. Sickness, however, recurred and by 10 p.m. he was pulseless. He died at midnight on the day of admission, death being due rather to cardiac failure than to intoxication.

The condition of the blood was very interesting. To the naked eye it shewed an obvious lipaemia, while the centrifugalised specimen gave a copious deposit of fat.

POST MORTEM: Heart: Fatty degeneration. Valves healthy.

Lungs: Acute bronchitis.

Pancreas: Some enlargement: very firm: the head presented a milky appearance below the capsule.

Kidneys: Subacute nephritis.



C. P. F. CALORIES

GRAMMES

DIET

Sept 28 d.

Age 8

Schoolboy.

Name D. Fisher Date Sept 28

ACETONE

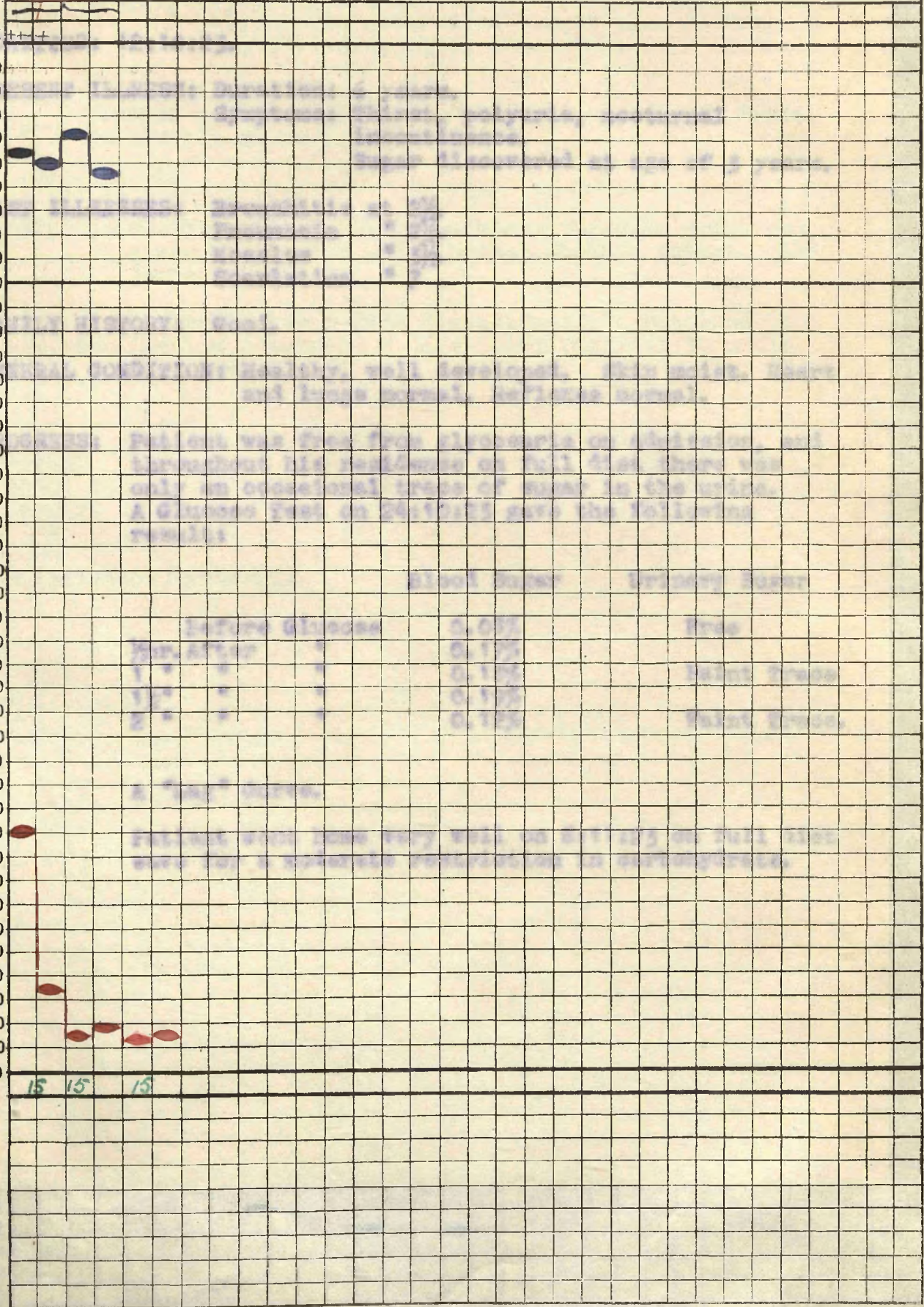
Age 0.45 420  
Ward 0.40 410  
Journal 0.35 400  
Page 0.30 390  
BLOOD 0.25 380  
SUGAR 0.20 370  
PER 0.15 360  
CENT 0.10 350  
0.05 340

TOTAL  
URINARY  
SUGAR

Date of Admission 28/9/23

INSULIN UNITS.

WEIGHT  
IN KILOS



J. F.

Age 8

Schoolboy.

ADMITTED: 12:10:23.

PRESENT ILLNESS: Duration: 6 years.

Symptoms: Thirst, polyuria, nocturnal  
incontinence.

Sugar discovered at age of 3 years.

PAST ILLNESSES: Bronchitis at  $2\frac{1}{2}$   
 Pneumonia "  $2\frac{1}{2}$   
 Measles "  $3\frac{1}{2}$   
 Scarlatina " 7.

FAMILY HISTORY: Good.

GENERAL CONDITION: Healthy, well developed. Skin moist. Heart  
 and lungs normal. Reflexes normal.

PROGRESS: Patient was free from glycosuria on admission, and  
 throughout his residence on full diet there was  
 only an occasional trace of sugar in the urine.  
 A Glucose Test on 24:10:23 gave the following  
 result:

				Blood Sugar	Urinary Sugar
	Before	Glucose		0.08%	Free
$\frac{1}{2}$ hr.	After	"	"	0.17%	
1 "	"	"	"	0.18%	Faint Trace
$1\frac{1}{2}$ "	"	"	"	0.19%	
2 "	"	"	"	0.12%	Faint Trace.

A "Lag" Curve.

Patient went home very well on 8:11:23 on full diet  
 save for a moderate restriction in carbohydrate.

Henry Shine

Age 42

Tailor

I. 16

ADMITTED: 17:10:23.

PRESENT ILLNESS:

Duration: 1 year.

Symptoms: Thirst, polyuria, weakness, loss of weight.

Glycosuria discovered 1 year ago. At work on modified diet until admission.

PAST ILLNESSES: Measles in childhood.

Vesical calculus 1915: operation: removal by Mr Parry.  
No trouble since.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Small man: thin-but appears healthy. Skin moist and clear. Heart and lungs normal. Abdomen normal. Knee jerks normal.

PROGRESS: Urine sugar free after 6 days diet: glycosuria returned on low diet. Insulin commenced 8 days later: required 30 units with a diet of 60:90:130 = 1770.

	Admission	Discharge
Weight	54 k	53 k
Blood Sugar	.395%	.200%
Urinary Sugar	90gms.	Free
& Acetone	++	-

A satisfactory result.







G.C.

Age 40

Lorryman.

ADMITTED: 7:11:23.

PRESENT ILLNESS: Duration: 2½ years.

Symptoms: Thirst, polyuria, frequency of micturition, loss of weight, weakness, dimness of vision, pain and swelling left leg. Sugar discovered 2 years ago: dieted with fair success.

PAST ILLNESSES: Whooping cough in childhood.

Measles " "

Gonorrhoea 1902.

Typhoid 1910.

Carbuncle Rt. Thigh 1910.

Tetanus, following sepsis Rt. foot 1912.

FAMILY HISTORY: Negative.

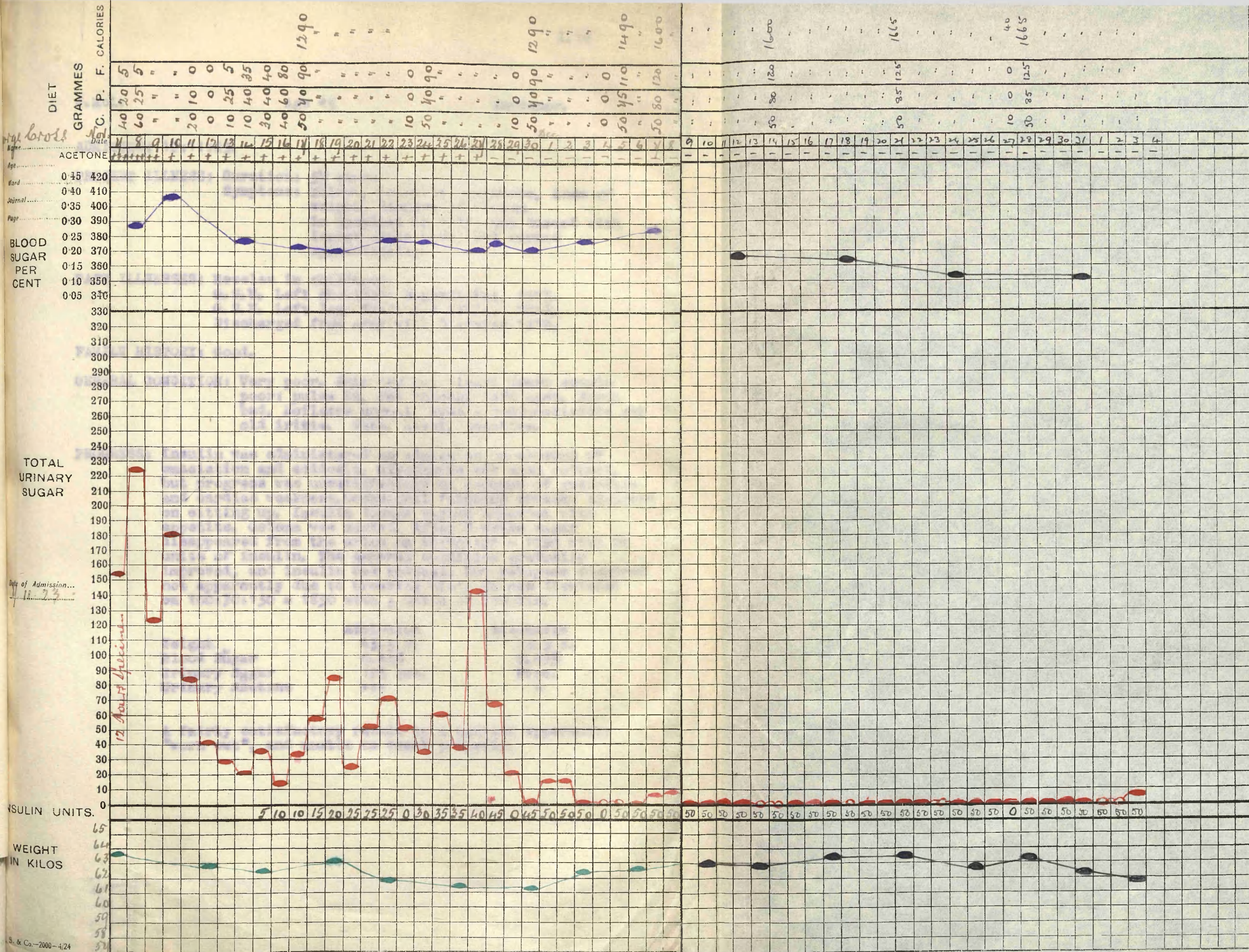
GENERAL CONDITION; Well nourished: muscles soft. Skin moist: scars Rt. thigh. Rt. sciatica. Deformity of chest-bulging Rt. lower ribs. Heart and lungs normal. Teeth carious: alveolar abscess. Reflexes normal. Wass. React. negative. Urine contained a cloud of albumen.

PROGRESS: Sugar and acetone were much reduced by diet, but could not be cleared entirely from the urine. Insulin was therefore commenced on the 8th. day and rapidly raised to 50 units. A trace of sugar remained on 50:80:120 = 1600, cleared by an occasional starve day. Patient left hospital free from symptoms on 50:85:125 = 1665 with 50 units of insulin. He was very stupid and occasionally broke diet.

	Admission	Discharge
Weight	63.5k.	61.8k.
Blood Sugar	0.24%	0.12%
Urinary Sugar	225gms.	Free
Urinary Acetone	+++	-

A satisfactory result, but prognosis was bad on account of poor intelligence.







J. McL.

Age 45

Gardener.

ADMITTED: 12:11:23.

PRESENT ILLNESS: Duration: 5½ years.

Symptoms: Thirst, polyuria, weakness, loss of weight, dimness of vision.

In hospital in 1918: has dieted with lapses since then, and worked spasmodically.

PAST ILLNESSES: Measles in childhood.

G.S.W. Left shoulder: superficial, 1917.

G.S.W. Left leg: fractured tibia, 1918.

Discharged from Army with Diabetes 1919.

FAMILY HISTORY: Good.

GENERAL CONDITION: Very poor. Skin dry but clear. Heart sounds poor: pulse 80. Few rhonchi left apex. Teeth bad. Reflexes normal. Eyes - conjunctivitis and old iritis. Wass. React. negative.

PROGRESS: Insulin was administered on admission on account of emaciation and acidosis. Glycosuria was much reduced, but progress was unsatisfactory on account of gastritis and cardiac weakness. Occasional fainting attacks occurred on sitting up. Insulin dosage varied somewhat with appetite. Oedema was marked. After 8 weeks sugar disappeared from the urine on 80:90:120 = 1760 with 20 units of insulin. The general condition gradually improved, and insulin was reduced: but relapses occurred not apparently due to breaking diet. He was dismissed on 100:70:130 = 1850 with 5 units of insulin.

	Admission	Discharge
Weight	43.5 k.	43.5 k.
Blood Sugar	0.40%	0.20%
Urinary Sugar	193 gms.	Free.
Urinary Acetone	+++	-

A fairly satisfactory result in a patient apparently "worn out", and unable to react properly.



[illegible]



ADMITTED: 15:11:23.

PRESENT ILLNESS:

Duration: 7 years.

Symptoms: frequency of micturition, polyuria, thirst, loss of weight.

14 days ago all ++, with headache and drowsiness. At work on diet till then. On day before admission passed 600 oz. of urine.

PAST ILLNESSES: Scarlet fever in childhood.

Thirst and polyuria 7 years ago: transient, passed off without diet.

FAMILY HISTORY: Mother suffers from T.B., family history otherwise negative.

GENERAL CONDITION: Emaciated, drowsy and listless, has to be roused to answer questions. Skin very dry. Heart normal: pulse 80, soft. Lungs normal: respirations 24, easy. Tongue dry: breath smells of acetone. Constipated. Reflexes normal.

PROGRESS: Insulin commenced at once on account of emaciation and acidosis. Collapsed shortly after admission - pulse 140. Improved under stimulation, but drowsiness very marked and difficulty in feeding. Three days later, after insulin 35 +30 +25 acidosis cleared: very bright and skin moist. Progress thereafter uninterrupted. Insulin reduced. Dismissed on diet 110:85:130 = 1950 with insulin 10 units.

	Admission	Discharge
Weight	31½ k.	34.8 k.
Blood Sugar	.48%	.10%
Urinary "	112gms.	Free.
" Acetone	++	-

A very satisfactory result.







Robert MacDonald

Age 32

Tramwayman

ADMITTED: 3:12:23  
 APRESENT ILLNESS:

Duration: 1 year.

Symptoms: Thirst, frequency of micturition, polyuria, loss of weight, easily fatigued; sugar discovered April 1923. Recent breathlessness on exertion. On moderately restricted diet and at work till admission.

PAST ILLNESSES: Mumps at 18

G.S.W. Rt Shoulder 1916:sepsis.

G.S.W. Left forearm 1918:amputation at elbow.

Influenza 1918.

FAMILY HISTORY: Negative.

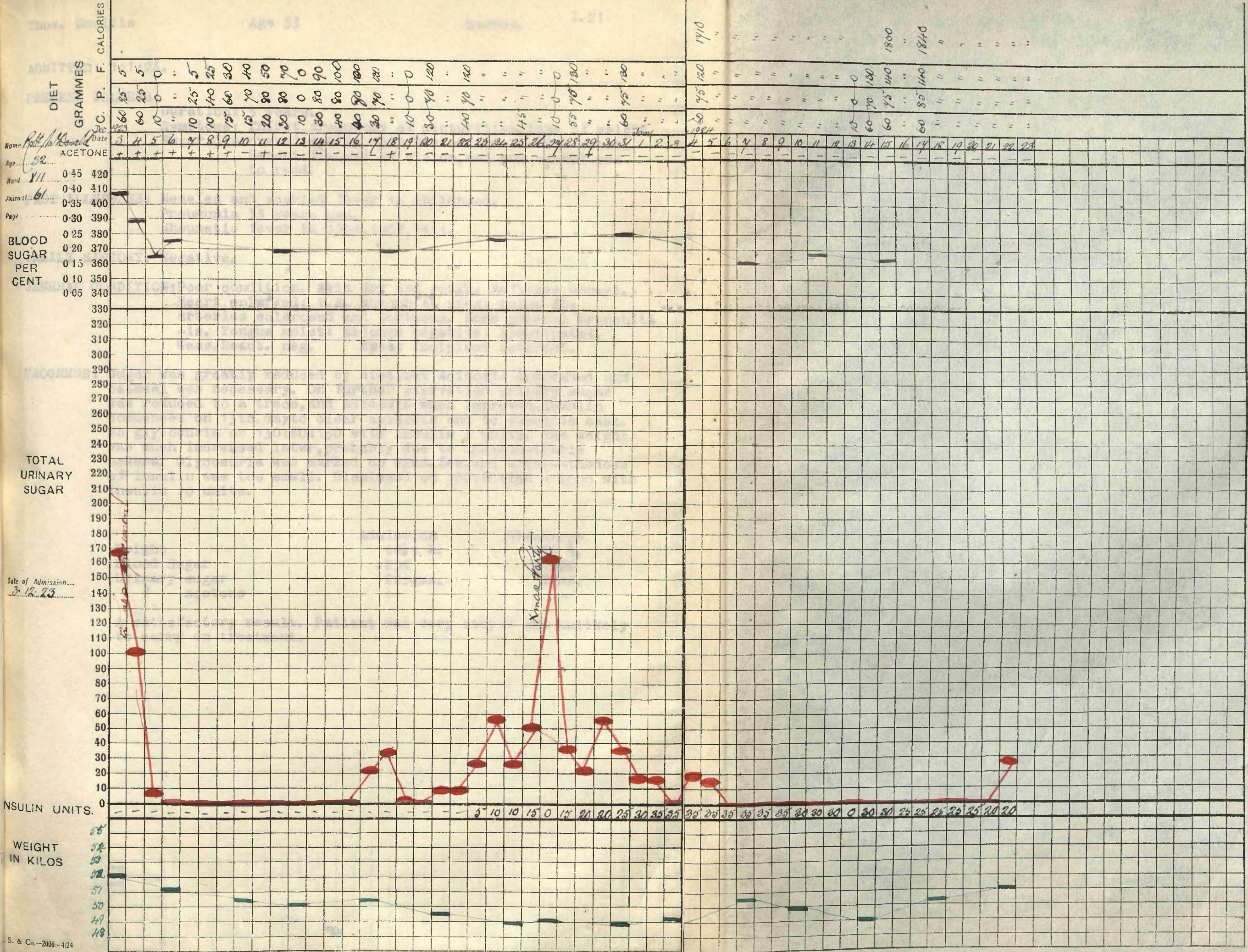
GENERAL CONDITION: Fair development:skin moist and clear:knee jerks and ankle jerks absent:pupils normal.Wasting of muscles of shoulder girdle.Heart lungs and abdomen normal.  
 Wass.React.neg.

PROGRESS:Attempt to clear glycosuria by diet unsuccessful,a trace persisting.Insulin commenced after three weeks,and raised to 35 units.A trace of sugar remained on 60:75:120=1710,but patient was certainly eating unauthorised food.Dismissed on 60:85:140 =1840,with insulin 20 units - a slightly reduced dose for safety.

	Admission	Discharge
Weight	52 k	51 k
Blood Sugar	.358%	.152%
Urinary Sugar	100gms.	Trace
" Acetone	+	-

A satisfactory result,but patient would not "play fair".  
 Prognosis unfavourable.







ADMITTED: 1:1:24.

**PRESENT ILLNESS:**

Duration: 3 years.

Symptoms: Thirst, frequency of micturition, loss of weight,

Sugar discovered spring 1922. Dieted and at work since.

Recent dimness of vision, and now cannot see to read.

**PAST ILLNESSES:** Measles and scarlet fever in childhood.

Pneumonia 35 years ago.

Rheumatic fever in 1903, 1908, 1911.

**FAMILY HISTORY:** Negative.

**GENERAL CONDITION:** Poor condition. Skin dry and rough. Reflexes normal.

Heart enlarged: V.S. murmur at apex: pulse 80:

arteries sclerosed and tortuous. Some general bronchitis.

-is. Tongue moist: abdomen negative: constipated.

Wass. React. neg. Eyes: Incipient cataract.

**PROGRESS:** Sugar was greatly reduced by diet, but acidosis increased and oatmeal was necessary. On further starvation urinary sugar was reduced to a trace, and acidosis much improved. Insulin commenced on 13th. day to clear acidosis and to expedite case. No glycosuria on 130:80:150 with insulin 5 units. The weight was much increased later, probably due to undemonstrable oedema. Glycosuria was marked on 22nd. Feb. and after--the dose of insulin was too small. Dismissed on 100:80:260 = 2090 with insulin 10 units.

	Admission	Discharge
Weight	68½ k.	66 k.
Blood Sugar	.25%	.158%
Urinary sugar	225gms.	Free.
acetone	++	-

A satisfactory result. Patient was very stupid and unlikely to carry on treatment.



DIET  
GRAMMES  
C. P. F. CALORIES

Name *Leo Amell*  
Age *53* Sex *M*  
Date *1.1.24*

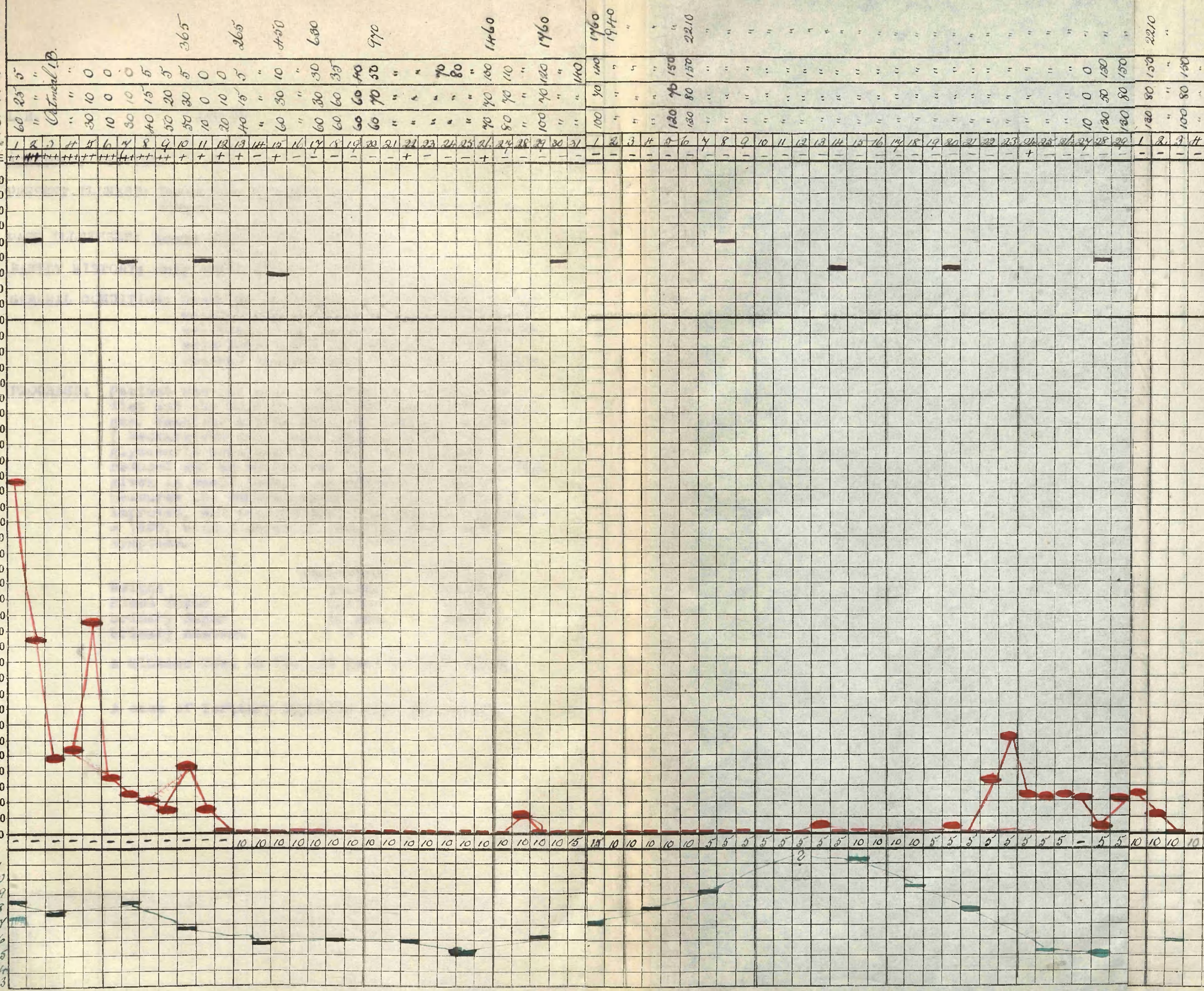
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0.45 420  
0.40 410  
0.35 400  
0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

TOTAL URINARY SUGAR

Date of Admission...  
*1.1.24*

INSULIN UNITS.

WEIGHT IN KILOS





L.H.

Age 60

Printer

ADMITTED: 16:1:24.

PRESENT ILLNESS: Duration: 15 years.

Symptoms: Nil save frequency of micturition.

PAST ILLNESSES: Acute Rheumatism 1919.

FAMILY HISTORY: Only child died at birth.

GENERAL CONDITION: Muscular and fairly well nourished. Skin moist. Ulcers left leg. Heart slightly enlarged: V.S. murmur at apex: arteries sclerosed. Left cataract and right retinal haemorrhage. Wass. React. +++ve.

PROGRESS: Patient was put upon a moderately restricted diet and the urinary sugar ran between 10 and 20 gms. Meantime active antisyphilitic remedies (Neokarsivan etc.) were administered. As glycosuria persisted the diet was slightly reduced and as weight was being lost insulin was given in small doses. As a result of these measures the general condition was greatly improved, and patient left hospital on 100:50:120 = 1680, with 5 units of insulin, free from symptoms.

	Admission	Discharge
Weight	53.4k.	53.8k.
Blood Sugar	0.18%	0.12%
Urinary Sugar	10 gms.	Free
Urinary Acetone	-	-

A Glucose Test on 18:1:24 gave a 'Lag' curve.

A case of Tertiary Syphilis with glycosuria.

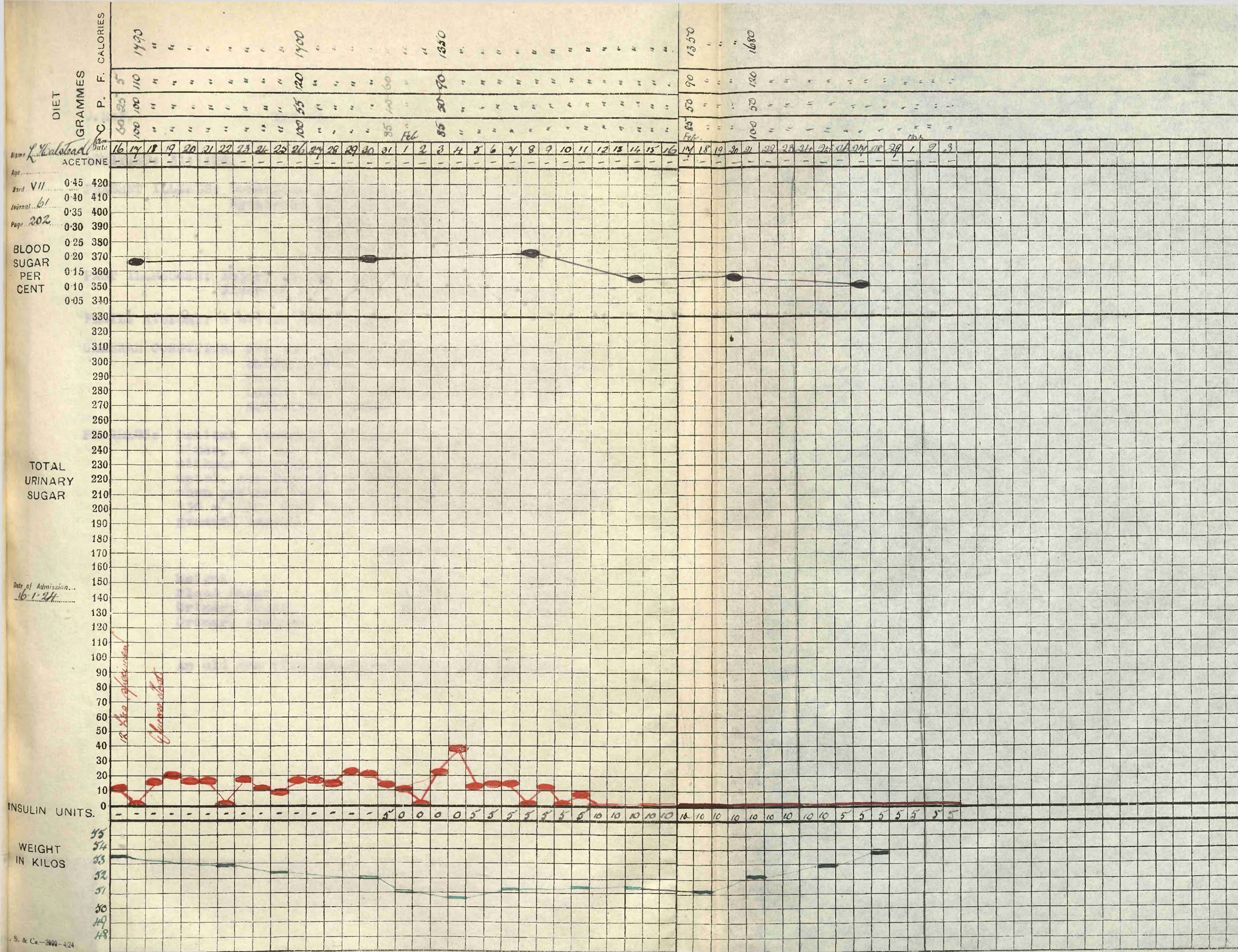


CALORIES

330

Date of Admission...  
16-1-24

WEIGHT  
IN KILOS





J. McF.

Age 64

Plumber.

ADMITTED: 31:1:24.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Thirst, polyuria, weakness, loss of weight, dimness of vision.

Sugar discovered 1916: carried on without diet.

PAST ILLNESSES: Injury to head and right knee 1910.

Ulcer on right great toe, result of injury 1923.

FAMILY HISTORY: A sister has diabetes.

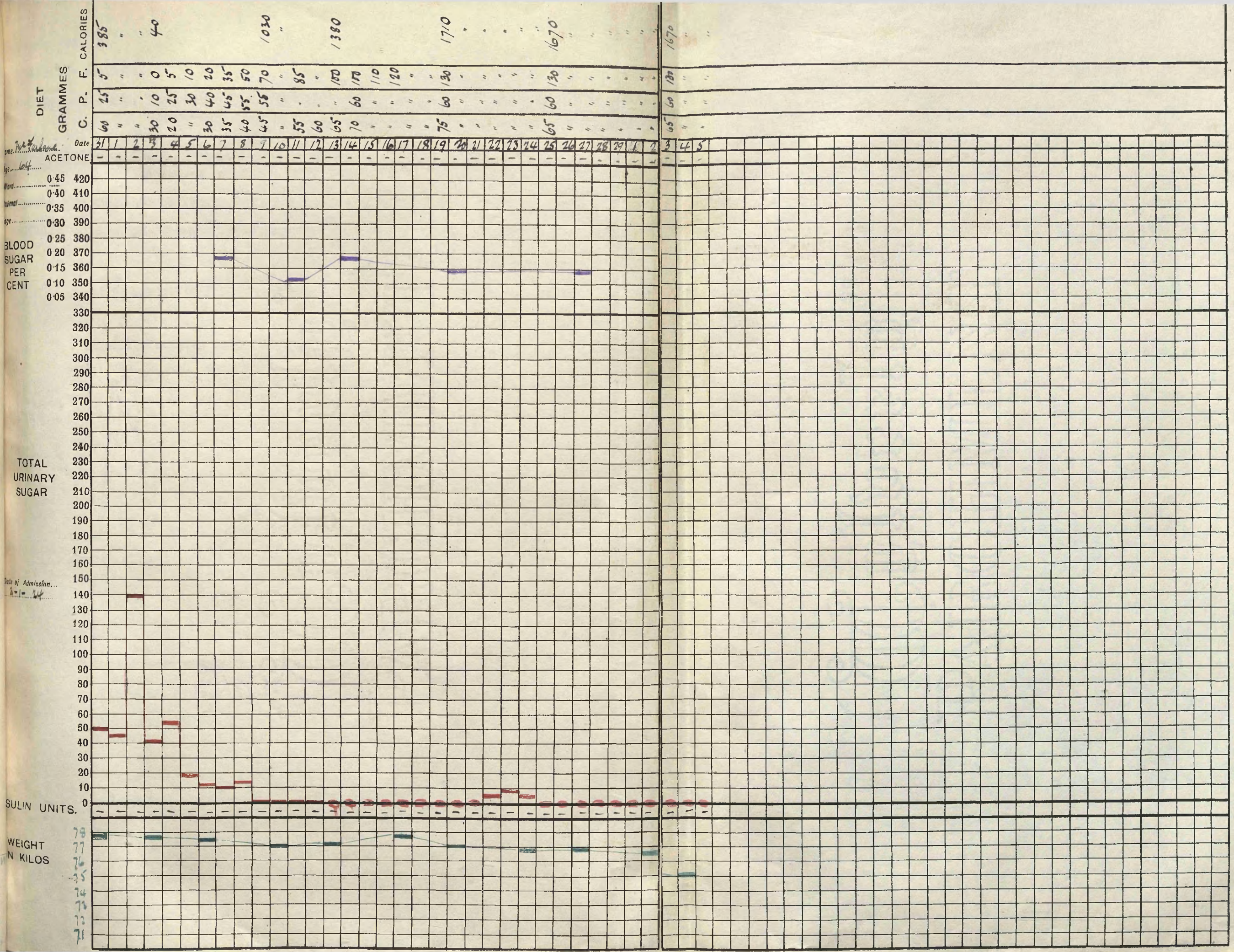
GENERAL CONDITION: Big and muscular but soft and flabby. Skin moist. Recent perineal abscess. Heart sounds poor: 2nd. aortic +. Arteries sclerosed. Lungs- some bronchitis. Tongue furred. Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic lines, and the urine was rendered sugar-free without starvation. Thereafter progress was uninterrupted, the glycosuria returning on one occasion only when patient broke diet. He left hospital on 65:60:130 = 1670, free from symptoms and much improved in general health.

	Admission	Discharge
Weight	77.8k.	76.5k.
Blood Sugar	0.158%	0.14%
Urinary Sugar	50gms.	Free
Urinary Acetone	-	-

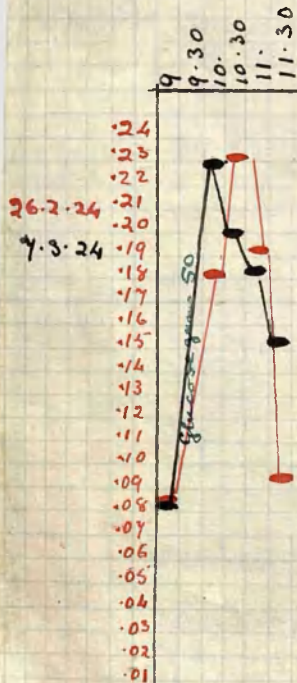
An old man with Arteriosclerosis and glycosuria.







Penman. vol. VII



James Penman

Age 31

Clerk.

ADMITTED: 6:2:24.

PRESENT ILLNESS: Duration: 1 month.

Symptoms: Thirst, frequency of micturition, loss of weight.  
Sugar discovered 4 weeks ago.

PAST ILLNESSES: Herpes Zoster 1905.

Alveolar Abscess 1920.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Height 5' 4½". Weight 11st. Well built: muscular. Skin moist and clear. Heart and lungs normal. Bowels regular. Reflexes normal. Eyes: high myopia, fundi and media normal.  
Wass. React. negative.

PROGRESS: The urinary sugar was rapidly reduced to a trace on 60:25:5., but as time was a consideration in this case, insulin was commenced on the 10th. day to expedite treatment. On 10 units with a moderately increased dietary a trace of sugar persisted. In view of this finding and the patient's excellent condition a Glucose Test was performed on 26:2:24 to confirm the diabetic nature of the case. The result was as follows:

	B. S.	U. S.
Pre. Gl.	.086%	2.6gms.
½ hr.	.182%	
1 hr.	.232%	5 gms.
1½ hr.	.191%	
2 hr.	.095%	10 gms.

Thereafter diet was fixed at 65:60:130 = 1670 , and without insulin the urine became sugar and acetone free and remained so. Save on admission the B. S. ran within normal limits. A second Glucose Test performed on 7:3:24 confirmed the previous finding.

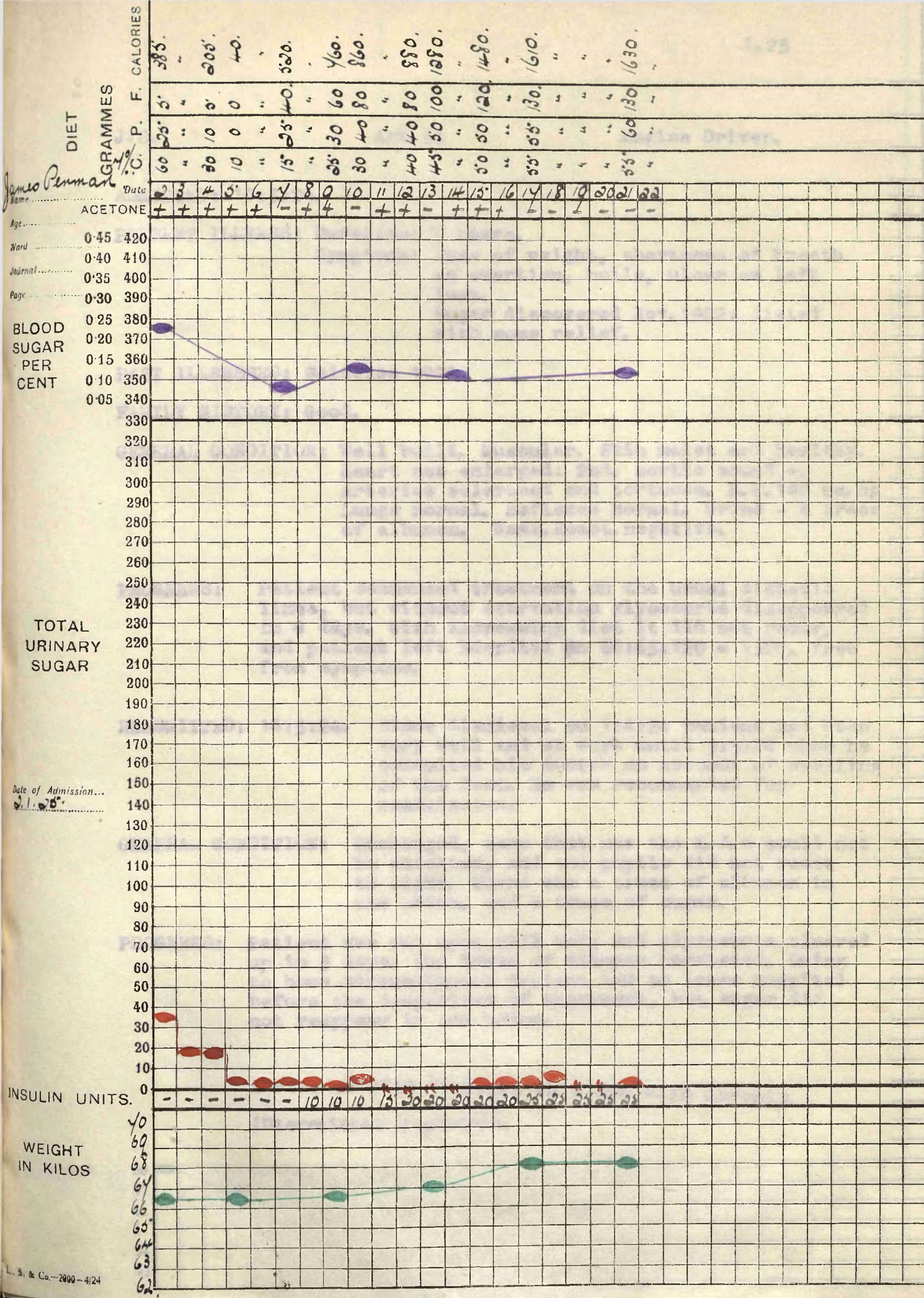
Apparently a diabetic, but Glucose Test shewed him to be in reality a "Lag" case. Advised to remain on restricted diet and to report progress.

READMITTED: 2:1:25. The general condition was unchanged. A Glucose Test now gave a typical Diabetic Curve, and patient required 25 units of insulin to render the urine sugar-free.









J. S.

Age 53

Engine Driver.

ADMITTED: 10:3:24.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Loss of weight, shortness of breath on exertion, boils, ulcer on left foot.

Sugar discovered Nov. 1922: dieted with some relief.

PAST ILLNESSES: Sciatica 1921.

FAMILY HISTORY: Good.

GENERAL CONDITION: Well built. Muscular. Skin moist and healthy. Heart not enlarged: 2nd. aortic sound +. Arteries sclerosed and tortuous. B.P. 140 mm. Hg. Lungs normal. Reflexes normal. Urine - a trace of albumen. Wass. React. negative.

PROGRESS: Patient commenced treatment on the usual dietetic lines, but without starvation glycosuria disappeared in 4 days. With increasing diet it did not recur, and patient left hospital on 60:65:120 = 1520, free from symptoms.

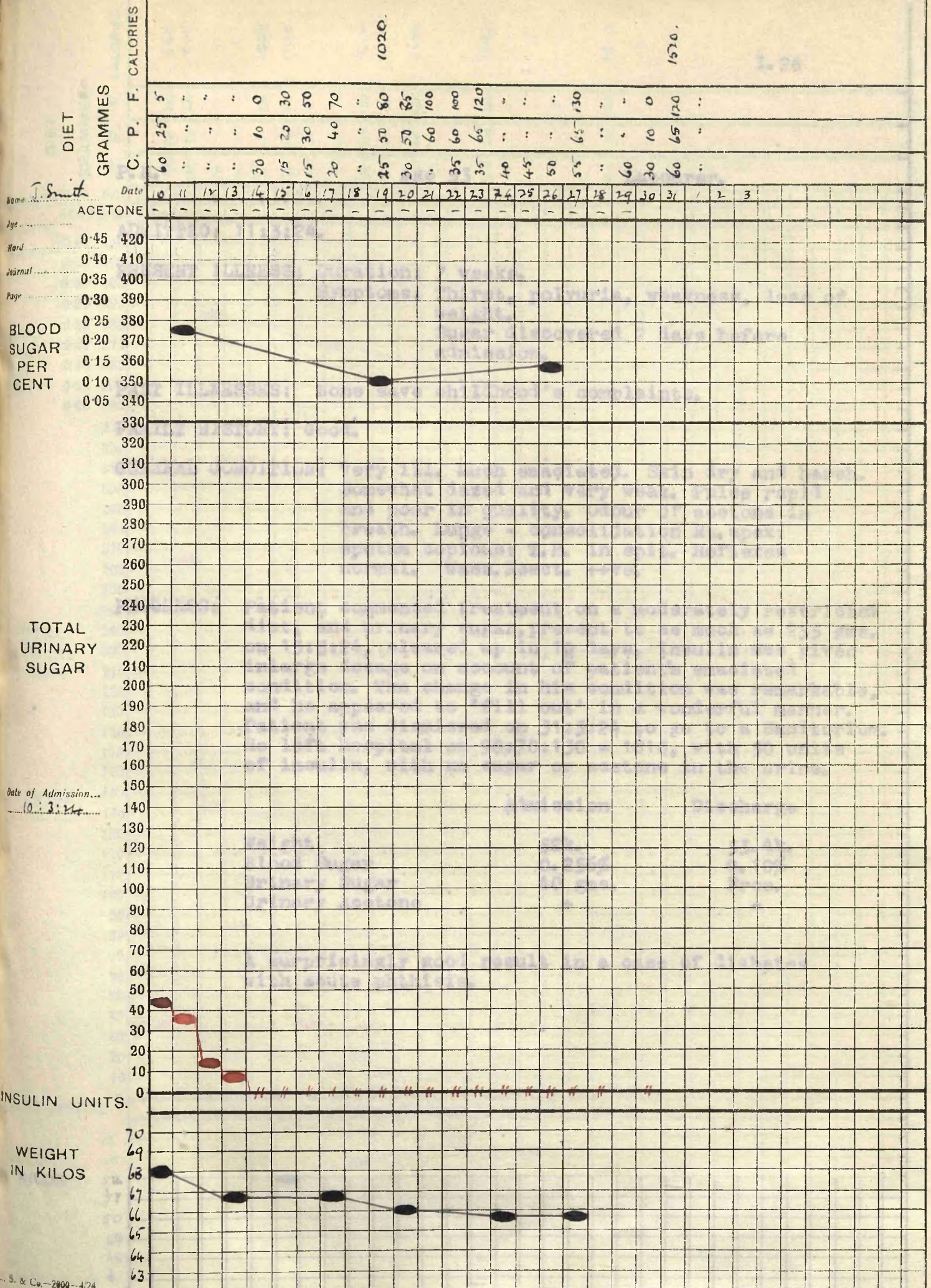
READMITTED: 14:5:24. Since dismissal on 1:4:24 patient had been very well and at work until 5:5:24 when he consulted his doctor on account of swelling of the feet. He was recommended for readmission.

GENERAL CONDITION: Unchanged, save that now the K.J.s could not be elicited, and the pupils did not react to light. There was a trace of albumen in the urine, and a trace of sugar.

PROGRESS: Patient was put upon milk only and glycosuria cleared up in 3 days. The trace of albumen persisted. Owing to home circumstances patient had to leave hospital before the completion of treatment, but sugar did not reappear in the urine.

A case of mild diabetes in a man with Chronic Interstitial Nephritis.





P.H.

Age 53

Labourer.

ADMITTED: 11:3:24.

PRESENT ILLNESS: Duration: 7 weeks.

Symptoms: Thirst, polyuria, weakness, loss of weight.

Sugar discovered 2 days before admission.

PAST ILLNESSES: None save childhood's complaints.

FAMILY HISTORY: Good.

GENERAL CONDITION: Very ill. Much emaciated. Skin dry and harsh. Somewhat dazed and very weak. Pulse rapid and poor in quality. Odour of acetone in breath. Lungs - consolidation Rt.apex: sputum copious: T.B. in spit. Reflexes normal. Wass.React. ++ve.

PROGRESS: Patient commenced treatment on a moderately restricted diet, and urinary sugar, present to as much as 255 gms. on 13:3:24, cleared up in 10 days. Insulin was given in large dosage on account of patient's emaciated condition. The change in his condition was remarkable, and he appeared to 'fill out' in a wonderful manner. Patient was dismissed on 31:3:24 to go to a Sanatorium. He left hospital on 90:70:130 = 1810, with 30 units of insulin, with no sugar or acetone in the urine.

	Admission	Discharge
Weight	52k.	53.4k.
Blood Sugar	0.256%	0.10%
Urinary Sugar	40 gms.	Free.
Urinary Acetone	+	-

A surprisingly good result in a case of diabetes with acute phthisis.





H.M.

Age 53

Brassfinisher.

ADMITTED: 11:5:24.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, loss of weight, weakness, shortness of breath on exertion.

Had been on diet for 6 months.

PAST ILLNESSES: Erysipelas 1894.

Pyorrhoea 1922: teeth extracted.

FAMILY HISTORY: Good.

GENERAL CONDITION: Fairly good. Looks his age. Muscles soft.

Skin dry but clear. Heart not enlarged:

sounds poor: arteries sclerosed and tortuous.

Lungs - slight emphysema. Tongue coated.

Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic lines, and without starvation the urine became sugar-free in a week. With increasing feeding, however, glycosuria returned, and, as the diet was then quite inadequate, insulin was begun. Thereafter progress was fairly satisfactory, but the urine could not be kept sugar-free owing to patient breaking diet. He left hospital on 50:65:120 = 1570, much improved.

	Admission	Discharge
Weight	59k.	60k.
Blood Sugar	0.355%	0.10%
Urinary Sugar	40 gms.	Free.
Urinary Acetone	-	-

A case of moderately severe diabetes in an unsatisfactory patient.



DIET

GRAMMES

Date

ACETONE

0.45

0.40

0.35

0.30

0.25

0.20

0.15

0.10

0.05

330

320

310

300

290

280

270

260

250

240

230

220

210

200

190

180

170

160

150

140

130

120

110

100

90

80

70

60

50

40

30

20

10

0

61

60

59

58

57

56

55

54

53

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PRESENT ILLNESS: Duration: 6 years.

Symptoms: Thirst, frequency of micturition, polyuria, loss of weight, weakness,

ADMITTED: 20:5:24.

Symptoms first in 1918. Sugar discovered in 1921 in Canada. Much improved on diet. Left Canada 1:5:24. stopped diet: headaches, giddiness, and marked loss of weight since that time.

PAST ILLNESSES: Always healthy.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Small:thin. Skin healthy. Heart normal:arteries palpable. Lungs - some bronchitis. Tongue furred:constipated. Knee jerks not elicited:pupils normal.

PROGRESS: Insulin was commenced at once, mainly on account of emaciation tho. acidosis was fairly marked. The dose was rapidly increased and acidosis had disappeared by the 4th. day. The morning B.S. was never above .16% , but great difficulty was experienced in bringing the urine sugar-free in spite of large doses of insulin. After six weeks on 30 units the urine became sugar-free and remained so on an equilibrium diet. He was dismissed on a diet of 60:75:135 = 1755. sugar and acetone free. The knee jerks were present on dismissal.

	Admission	Discharge
Weight	44.5k.	48.2k.
Blood Sugar	.89%	.07%
Urinary Sugar	5gms.	Free.
" Acetone	+	-

A difficult case with satisfactory result. Prognosis fair.







A. McM.

Age 49

Foreman Shipwright.

ADMITTED: 30:5:24.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Thirst, loss of weight, occasional sickness.

Sugar discovered April 1924: dieted for 3 weeks.

PAST ILLNESSES: Scarlet Fever in childhood.

FAMILY HISTORY: Good.

GENERAL CONDITION: Thin, but well developed. Heart and lungs normal. Some pyorrhoea : constipated.  
Reflexes normal.

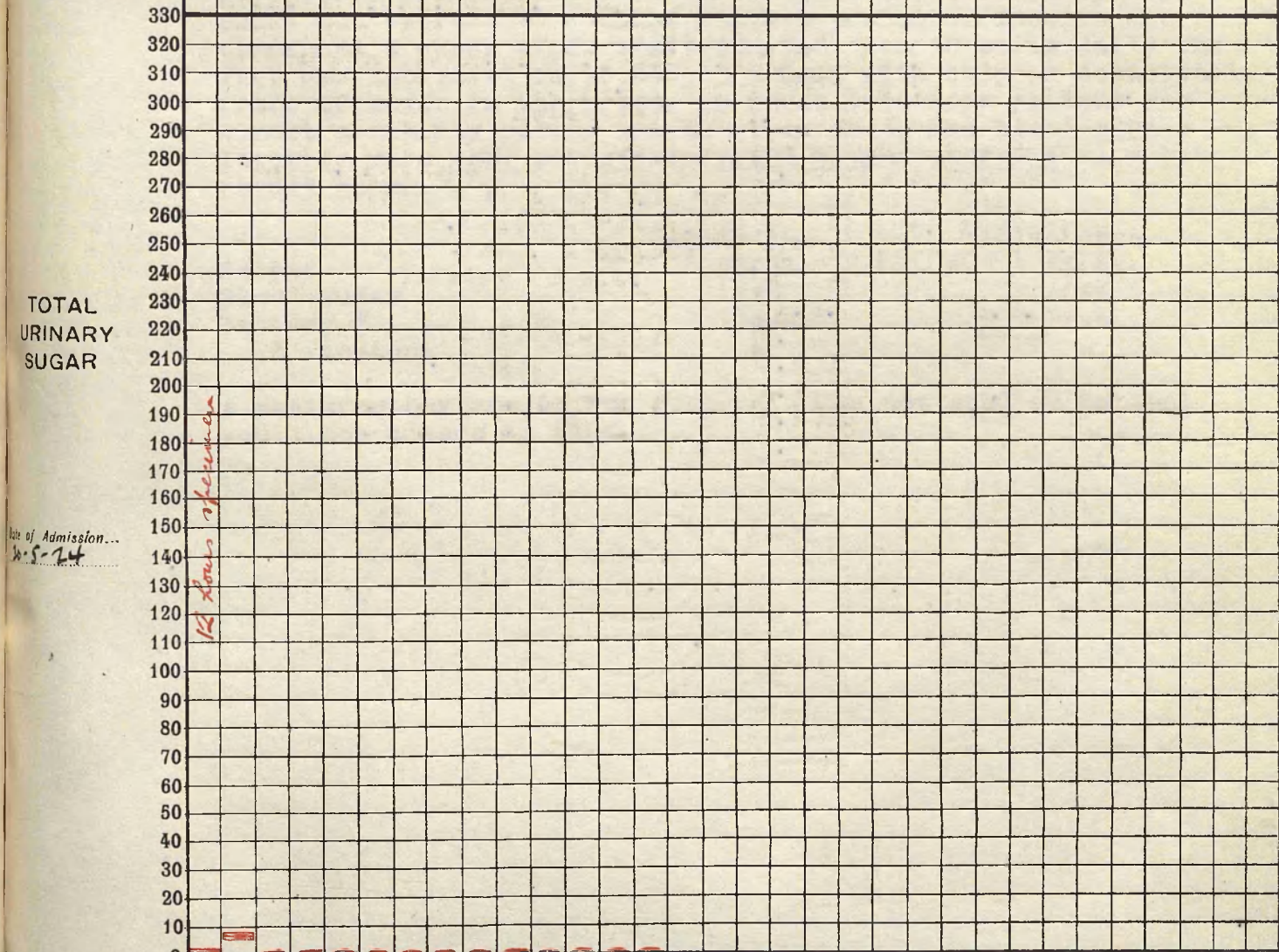
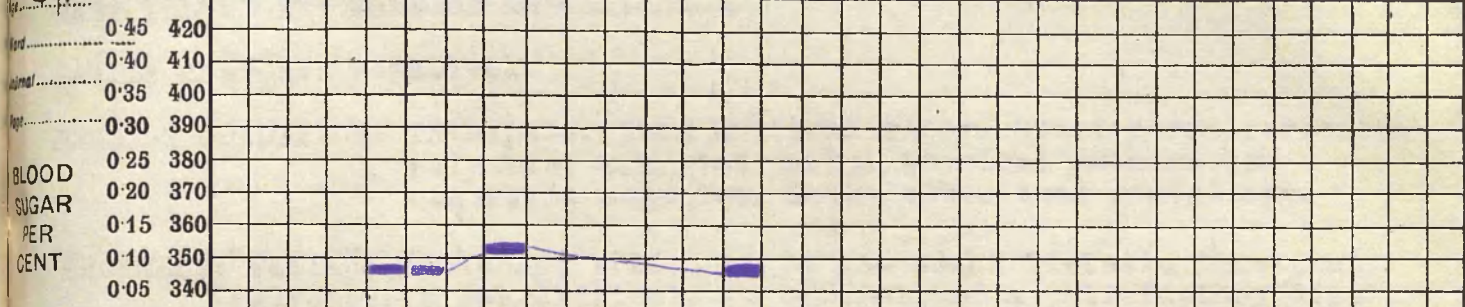
PROGRESS: Patient was given 200 gms. carbohydrate on admission in his diet and the urine contained only a trace of sugar. With a reduction to 100 gms. the urine became sugar-free. A Glucose Test was performed with the following result:

			B. S.	U. S.
	Before	Glucose	0.081%	Free
1/2 hr.	After	"	0.131%	
1 "	"	"	0.177%	1.2 gms
1 1/2 "	"	"	<del>0.082%</del>	
2 "	"	"	0.080%	Free

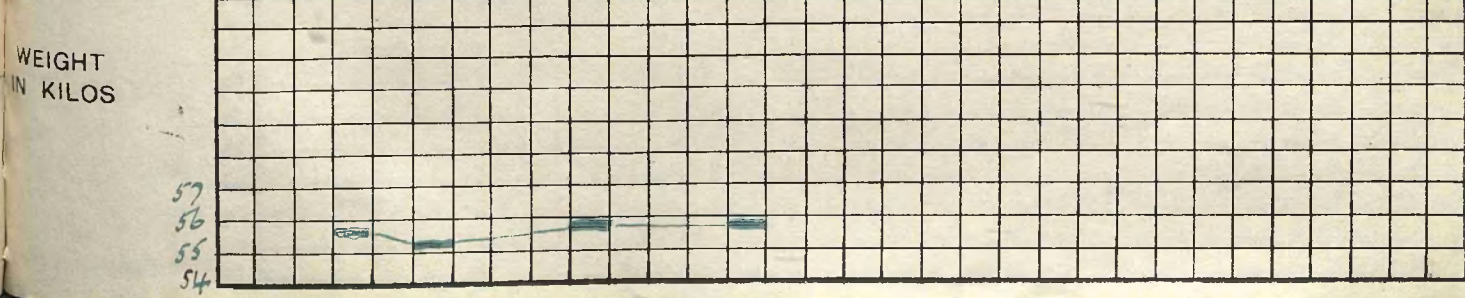
As this was obviously a 'Renal' curve patient was permitted to eat ordinary diet less obvious sugar. He was dismissed on a diet on these lines - 120:100:100 = 1780. The morning blood sugar reading during his residence was always within normal limits.

DIET	GRAMMES	C.	P.	F.	CALORIES
		200	100	100	2100
		100	100	100	1700
		30	10	0	520
		100	100	100	1700
		"	"	"	"
		"	"	"	"
		120	100	100	

Date	30	31	1	2	3	4	5	6	7	8	9	10	11	12
ACETONE	-	-	-	-	-	-	-	-	-	-	-	-	-	-



INSULIN UNITS.	30	31	1	2	3	4	5	6	7	8	9	10	11	12
	-	-	-	-	-	-	-	-	-	-	-	-	-	-



Name *A. McMillan*  
 49  
 BLOOD SUGAR PER CENT  
 TOTAL URINARY SUGAR  
 Date of Admission... *20-5-24*

12 hours specimen

57  
 56  
 55  
 54

John Cooper

Age 48

I. 30  
Ironmoulder.

ADMITTED: 24:6:24.

PRESENT ILLNESS: Duration: 2 weeks.

Symptoms: Thirst, polyuria, loss of weight. Sugar  
discovered 4 days ago.

PAST ILLNESSES: Measles in childhood.

FAMILY HISTORY: Negative.

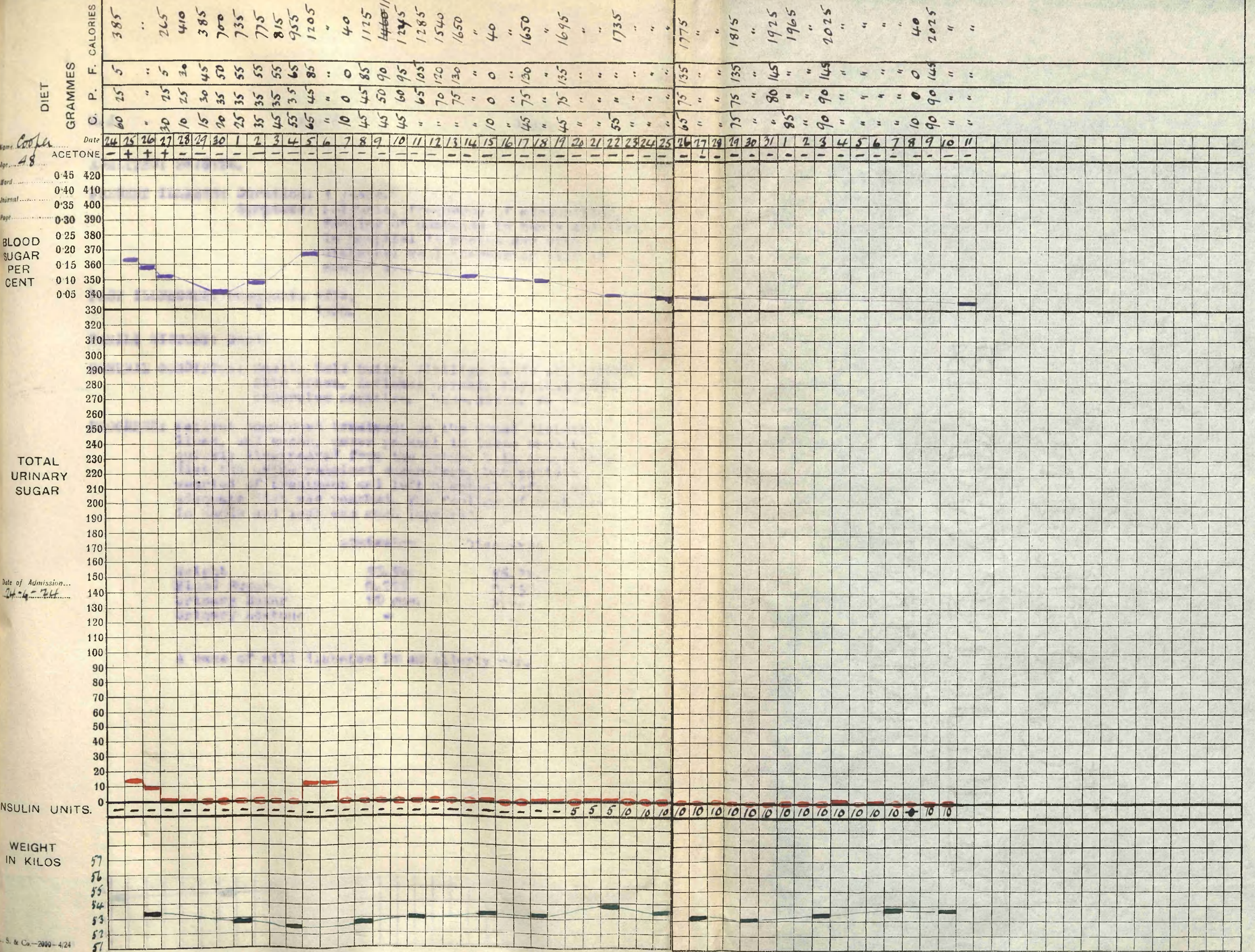
PRESENT CONDITION: Thin: pale. Skin moist and clear. Heart normal: arteries  
palpable: B.P. 140 mm. Hg. Physical examination  
otherwise negative. Looks older than stated age.

PROGRESS: Patient commenced treatment on the usual dietetic lines, but  
glycosuria was difficult to control, and a satisfactory diet  
could not be reached without its reappearance. Insulin was  
commenced 3 weeks after admission, and with 10 units daily the  
diet was increased to 90:90:145 = 2025 with only an occasional  
trace of sugar in the urine. On these occasions patient was  
almost certainly eating unauthorised food. The blood-sugars  
latterly were very satisfactory, and weight shewed a slow but  
steady gain.

	Admission	Discharge
Weight	53.3k.	53.8k.
Blood Sugar	.153%	.08%
Urinary "	15gms.	Free.
" Acetone	+	-

A satisfactory result, but prognosis was not good as patient  
would not adhere to diet.







H. J.

Age 64

Dyer.

ADMITTED: 24:6:24.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Polyuria, frequency of micturition,  
feeling of numbness in hands and legs.  
In hospital 18 months ago with  
diabetes: well thereafter till 12  
months ago.

PAST ILLNESSES: Pneumonia 1874.  
" 1920.

FAMILY HISTORY: Good.

GENERAL CONDITION: Small. Well built. Vitiligo hands and wrists.  
Skin moist. Reflexes normal. Physical exam.  
otherwise negative. Wass.React. ++ ve.

PROGRESS: Patient commenced treatment on the usual dietetic  
lines, and sugar, never present in large amount,  
quickly disappeared from the urine. With increasing  
diet the urine remained sugar-free, but patient  
wearied of treatment and left hospital before an  
adequate diet was reached. The feeling of numbness  
in hands and legs was much improved.

	Admission	Discharge
Weight	55.5k.	56.7k.
Blood Sugar	0.20%	0.15%
Urinary Sugar	10 gms.	Free
Urinary Acetone	-	-

A case of mild diabetes in an elderly man.

DIET		GRAMMES		C. P. F. CALORIES	
Name	Date	C.	P.	F.	Calories
H.J.	24	60	25	5	385
	25	"	"	"	"
	26	"	"	"	"
	27	30	25	5	410
	28	10	25	30	585
	29	15	30	45	715
	30	20	35	55	740
	1	25	35	55	"
	2	"	"	"	795
	3	35	35	55	835
	4	45	35	55	"
	5	"	"	"	955
	6	55	35	65	1205
	7	65	45	85	1580
	10	75	50	90	1705
	11	85	60	95	

ACETONE	
Date	Value
24	0.45
25	0.40
26	0.35
27	0.30
28	0.25
29	0.20
30	0.15
1	0.10
2	0.05
3	
4	
5	
6	
7	
10	
11	

BLOOD SUGAR PER CENT	
Date	Value
24	0.45
25	0.40
26	0.35
27	0.30
28	0.25
29	0.20
30	0.15
1	0.10
2	0.05
3	
4	
5	
6	
7	
10	
11	

TOTAL URINARY SUGAR	
Date	Value
24	0.45
25	0.40
26	0.35
27	0.30
28	0.25
29	0.20
30	0.15
1	0.10
2	0.05
3	
4	
5	
6	
7	
10	
11	

INSULIN UNITS.	
Date	Value
24	0.45
25	0.40
26	0.35
27	0.30
28	0.25
29	0.20
30	0.15
1	0.10
2	0.05
3	
4	
5	
6	
7	
10	
11	

WEIGHT IN KILOS	
Date	Value
24	55.5
25	55.5
26	55.5
27	55.5
28	55.5
29	55.5
30	55.5
1	55.5
2	55.5
3	55.5
4	55.5
5	55.5
6	55.5
7	55.5
10	55.5
11	55.5

A. B.

Age 36

Draughtsman

ADMITTED: 19:7:24.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Frequency of micturition, polyuria,  
pain in back.

Sugar discovered 6 weeks ago.

PAST ILLNESSES: Scarlet fever in childhood.

FAMILY HISTORY: Good.

GENERAL CONDITION: Well built: Well developed. Skin dry. No  
skin lesions. Reflexes normal. Tongue  
coated. Physical examination otherwise  
negative. Wass. React. neg.

PROGRESS: Patient commenced treatment on the usual dietetic  
lines, but starvation was never necessary as sugar  
was never present in the urine save as a trace.  
With increasing diet the carbohydrate reached 120gms.  
before glycosuria recurred. Acidosis was never  
troublesome. Patient left hospital on 100:80:145 =  
2025, free from symptoms.

	Admission	Discharge
Weight		
Blood Sugar	0.08%	
Urinary Sugar	Trace	Free
Urinary Acetone	-	+

A case of mild diabetes. A Glucose Test in this  
case gave a 'Diabetic' curve.



Name *Arthur Burley*  
Age *36 yrs*

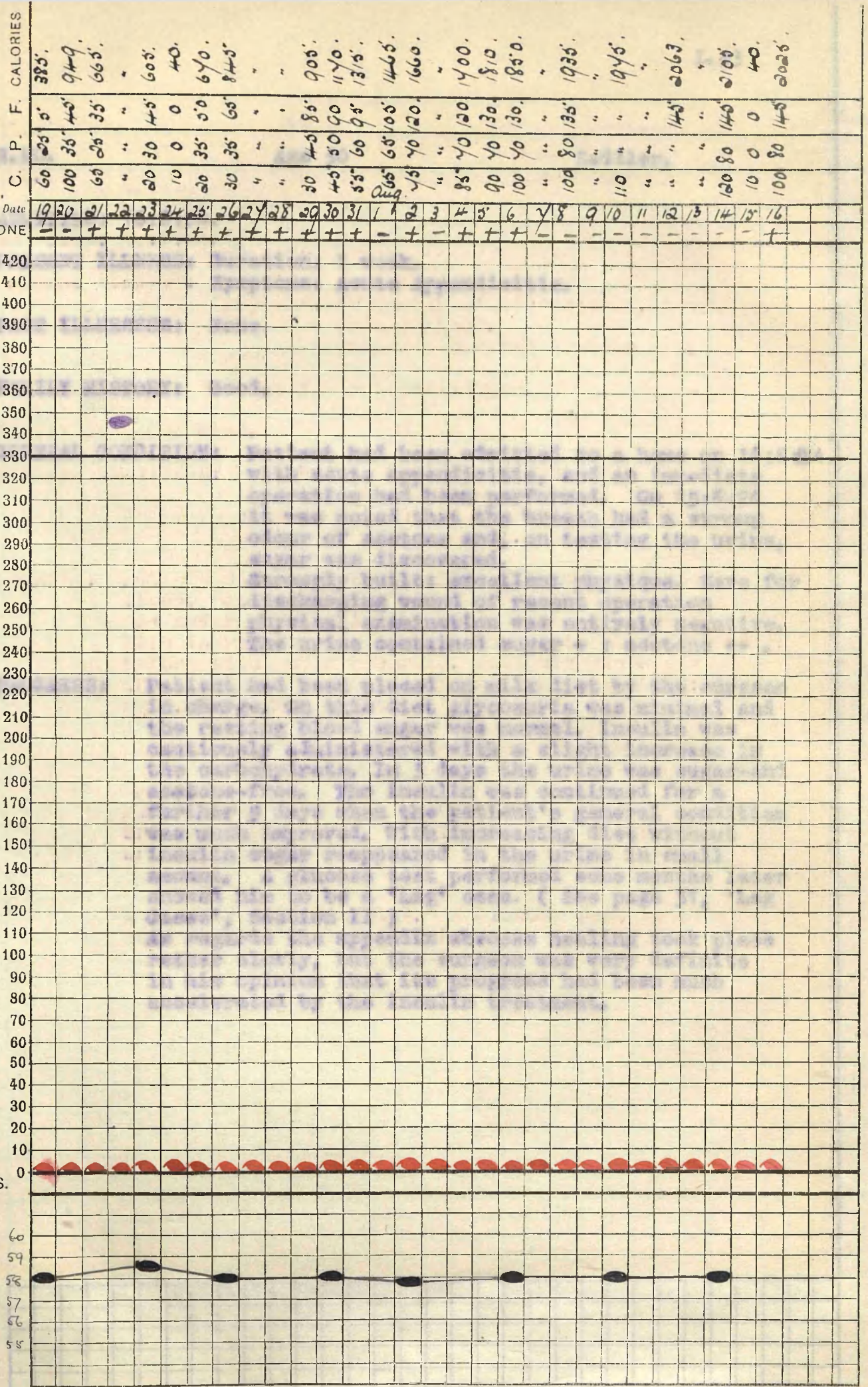
Ward *4*  
Journal  
Page  
BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

Date of Admission... *10/4/24*

INSULIN UNITS.

WEIGHT IN KILOS



N. NO.

Age 30

Saddler.

ADMITTED: 16:8:24.

PRESENT ILLNESS: Duration: 1 week.  
Symptoms: Acute Appendicitis.

PAST ILLNESSES: None.

FAMILY HISTORY: Good.

GENERAL CONDITION: Patient had been admitted to a home on 14:8:24 with acute appendicitis, and an immediate operation had been performed. On 15:8:24 it was noted that the breath had a strong odour of acetone and, on testing the urine, sugar was discovered. Strongly built: excellent physique. Save for discharging wound of recent operation physical examination was entirely negative. The urine contained sugar + : acetone ++ .

PROGRESS: Patient had been placed on milk diet by the surgeon in charge. On this diet glycosuria was minimal and the resting blood sugar was normal. Insulin was cautiously administered with a slight increase in the carbohydrate. In 3 days the urine was sugar-and acetone-free. The insulin was continued for a further 5 days when the patient's general condition was much improved. With increasing diet without insulin sugar reappeared in the urine in small amount. A glucose test performed some months later showed him to be a 'Lag' case. ( See page 31, 'Lag Cases', Section II )  
As regards the appendix abscess healing took place rather slowly, but the surgeon was very definite in his opinion that its progress had been much accelerated by the insulin treatment.





T.O.

Age 54

Gamekeeper.

ADMITTED: 23:8:24.

PRESENT ILLNESS: Duration: 4 years.

Symptoms: Thirst, voracious appetite, general weakness.

Sugar discovered 1920 when in hospital for operation on leg. Dieted with improvement. Carbuncle Aug. 1923: increasing weakness since.

PAST ILLNESSES: Chill 1915.

Accident to left leg: incised wound involving muscle 1920.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well built: florid. Skin moist. Scar of old wound left leg above knee: walks with slight limp. Heart and lungs normal. Tongue furred: constipated. Reflexes normal.

Urine - trace of albumen : no casts.

PROGRESS: Entirely satisfactory. On a falling dietary sugar disappeared from the urine in 3 days, and never recurred during the subsequent period of treatment. The albuminuria quickly cleared up, and acetone was never present. Patient was dismissed on 90:80:120 = 1760, free from symptoms.

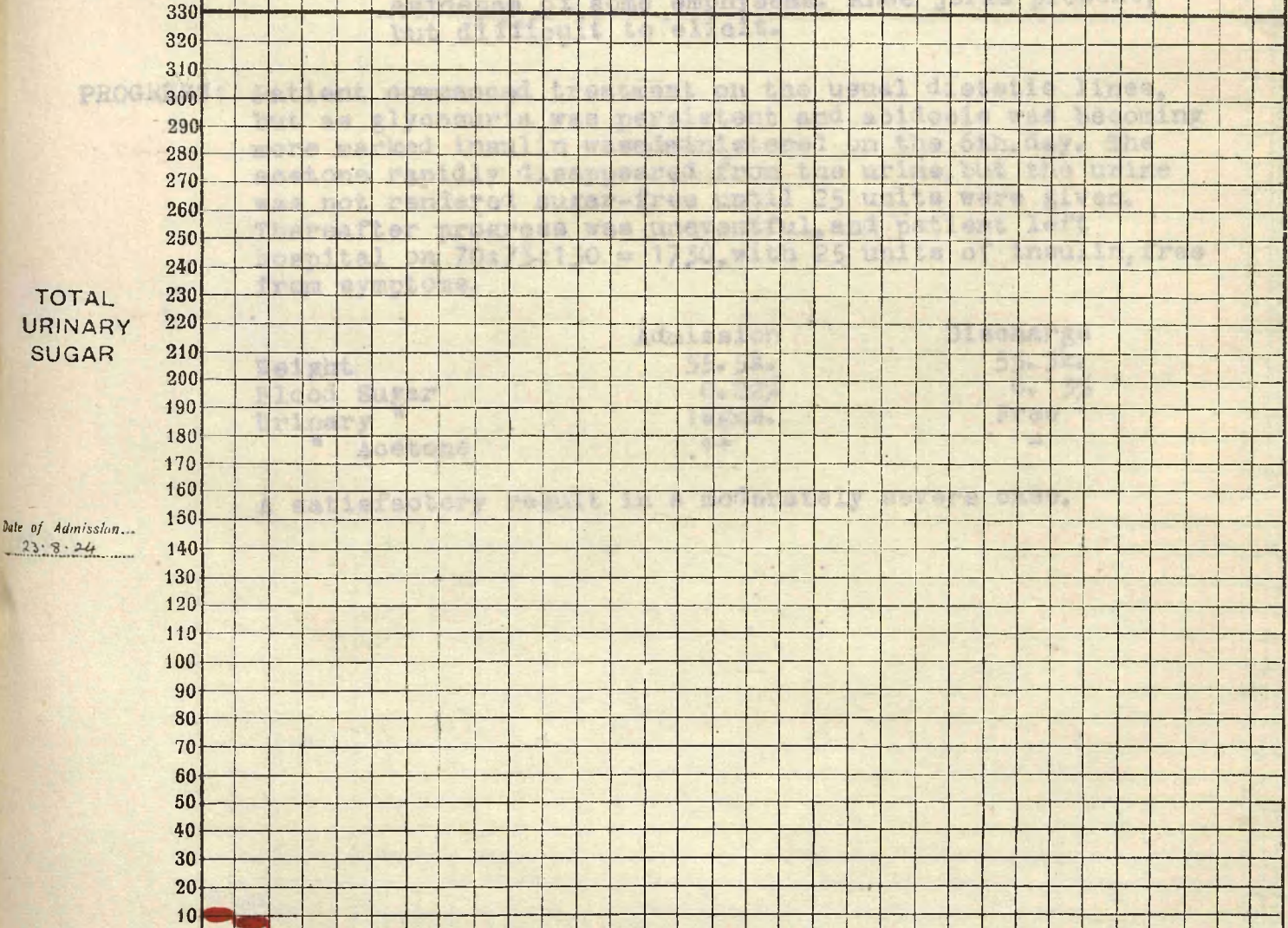
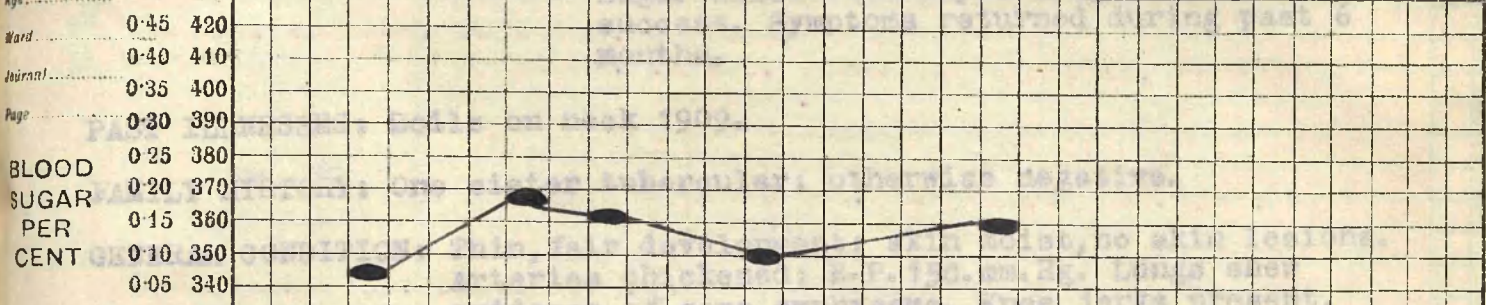
	Admission	Discharge
Weight	82.5k.	81.5k.
Blood Sugar	0.08%	0.15%
Urinary Sugar	10gms.	Free
Urinary Acetone	-	-

A satisfactory result in a case of mild diabetes.

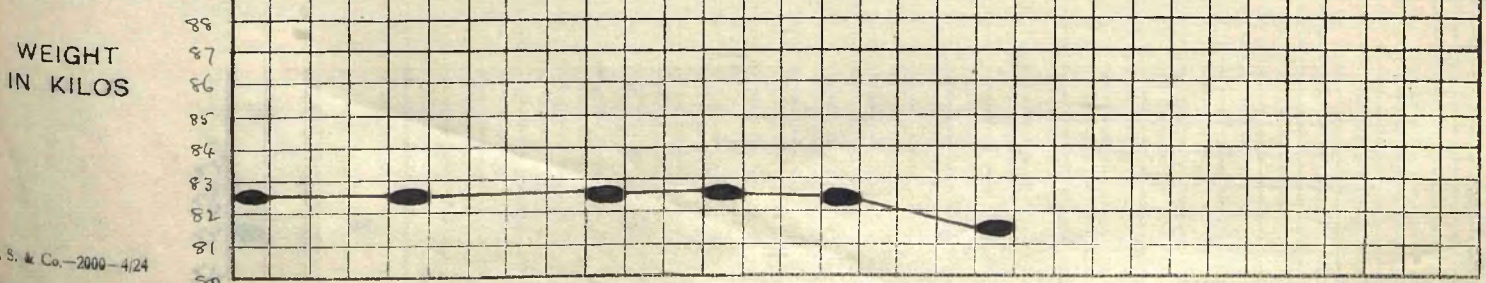


DIET	GRAMMES	C.	P.	F.	CALORIES
		60	25	5	385
		30	10	0	160
		15	15	30	390
		25	30	40	540
		35	30	50	730
		45	35	65	805
		55	45	85	1005
		65	45	85	1045
		75	50	90	1310
		85	60	95	1435
		90	65	100	1520
		90	70	110	1630
		90	70	110	1740
		90	75	120	1760
		90	80	120	1760

Name: George Date: 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12



INSULIN UNITS.



ADMITTED: 23:8:24.

## PRESENT ILLNESS:

Duration: 2 years.

Symptoms: Thirst, polyuria, loss of weight, weakness in legs.

Sugar discovered 2 years ago: dieted with success. Symptoms returned during past 6 months.

PAST ILLNESSES: Boils on neck 1909.

FAMILY HISTORY: One sister tubercular: otherwise negative.

GENERAL CONDITION: Thin, fair development: skin moist, no skin lesions. Arteries thickened: B.P. 150. mm. Hg. Lungs shew evidence of some emphysema. Knee jerks present, but difficult to elicit.

PROGRESS: Patient commenced treatment on the usual dietetic lines, but as glycosuria was persistent and acidosis was becoming more marked insulin was administered on the 6th. day. The acetone rapidly disappeared from the urine, but the urine was not rendered sugar-free until 25 units were given. Thereafter progress was uneventful, and patient left hospital on 70:75:130 = 1730, with 25 units of insulin, free from symptoms.

	Admission	Discharge
Weight	55.5k.	55.5k.
Blood Sugar	0.22%	0.13%
Urinary "	18gms.	Free
" Acetone	++	-

A satisfactory result in a moderately severe case.



Name *Wm Cunningham*

Age *44 yrs*

Ward *70*

Journal *63*

Page

BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

Date of Admission... *28/8/24*

INSULIN UNITS.

WEIGHT IN KILOS

DIET

GRAMMES

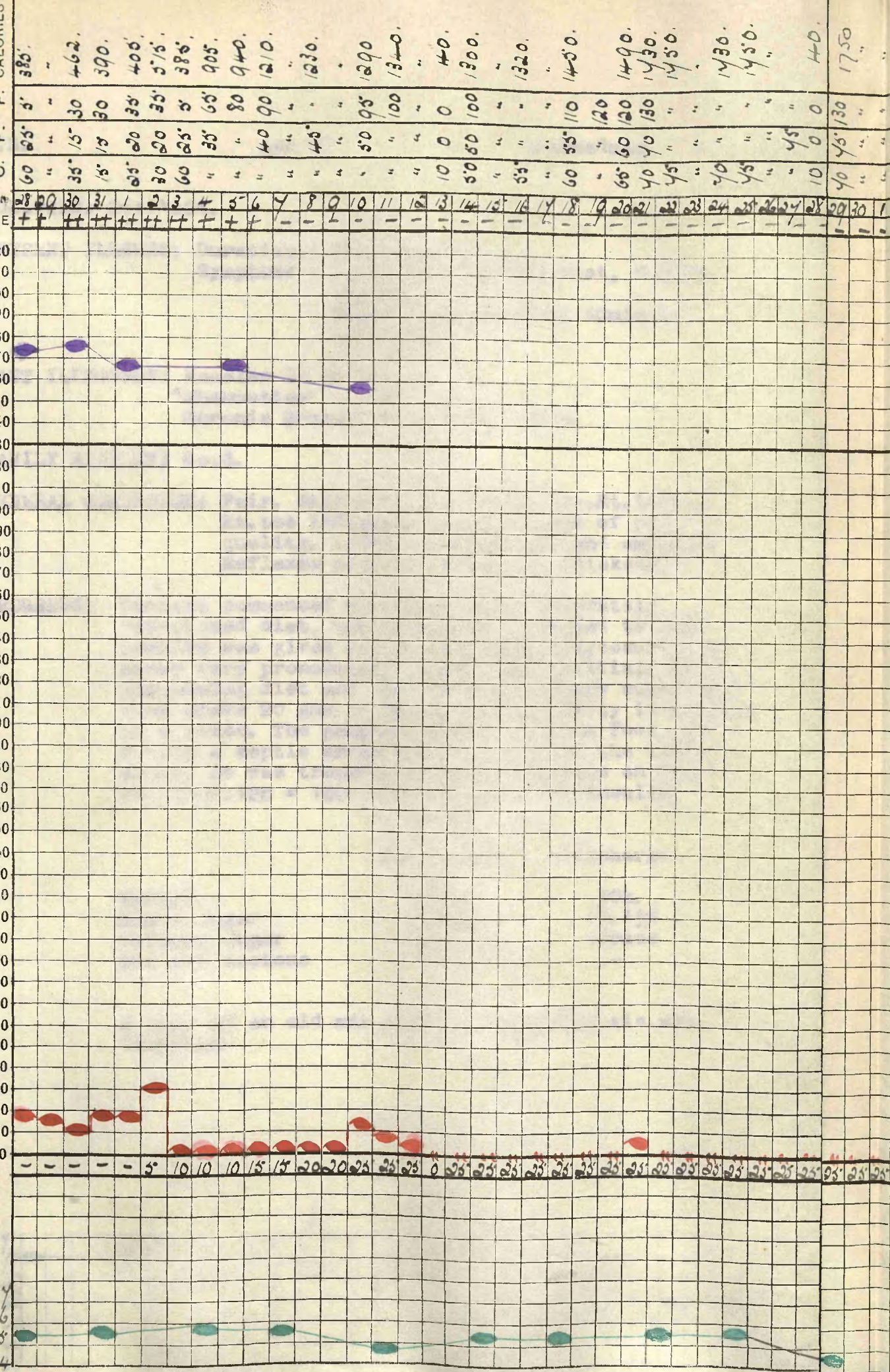
C. P. F. CALORIES

CETONE

0.45  
0.40  
0.35  
0.30  
0.25  
0.20  
0.15  
0.10  
0.05

330  
320  
310  
300  
290  
280  
270  
260  
250  
240  
230  
220  
210  
200  
190  
180  
170  
160  
150  
140  
130  
120  
110  
100  
90  
80  
70  
60  
50  
40  
30  
20  
10  
0

5.7  
5.6  
5.5  
5.4





M.M.

Age 68

Pitheadman.

ADMITTED: 26:11:24.

PRESENT ILLNESS: Duration : Unknown.

Symptoms : Gangrene of Right foot, following injury.

Sugar discovered on admission to hospital.

PAST ILLNESSES: Measles in childhood.

'Rheumatism' 1919.

Chronic Bronchitis - some years.

FAMILY HISTORY; Good.

GENERAL CONDITION: Fair. Skin soft. Gangrene 2nd. Rt. toe: 3rd. Rt. toe inflamed. Heart sounds of poor quality. Lungs - bronchitis and emphysema. Reflexes normal. Arteries thickened.

PROGRESS: Patient commenced treatment on a moderately restricted diet, but as gangrene tended to spread insulin was given on the 4th. day. Glycosuria, never very pronounced, soon became trivial. With increasing diet and insulin the urinary sugar never rose above 20 gms. in 24hrs, and latterly it was free to a trace. The gangrene spread to the foot and on 2:1:25 a septic arthritis occurred in the left elbow. He was transferred for operation on 7:1:25 on 70:60:120 = 1600 with 45 units of insulin.

	Admission	Discharge
Weight	70k.	70k.
Blood Sugar	0.25%	0.15%
Urinary Sugar	30gms.	Trace
Urinary Acetone	+	-

A case of an old man with Arteriosclerosis and Diabetes.







A. H.

Age 51

Boilermaker.

ADMITTED: 16:12:24.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Thirst, polyuria, loss of weight, weakness, dimness of vision.

Sugar discovered May 1922: dieted with fair success.

PAST ILLNESSES: Measles in childhood.

FAMILY HISTORY: Good.

GENERAL CONDITION: Well built. Skin moist. Varicose veins both legs. Heart and lungs normal. Tongue coated: odour of acetone in breath. Reflexes normal.

PROGRESS: Patient commenced treatment on the routine dietetic lines, but he showed such evidence of acidosis that insulin was administered on the 3rd. day. Despite rapidly increasing dosage difficulty was experienced in keeping the urine sugar-free, but latterly this was accomplished with 55 units. Patient left hospital on 60:55:130 = 1630, free from symptoms and with his eyesight much improved.

	Admission	Discharge
Weight	65.5k.	66k.
Blood Sugar	0.25%	0.12%
Urinary Sugar	144 gms.	Free
Urinary Acetone	+++	-

A satisfactory result with insulin in a case of severe diabetes.







Mrs. McD.

Age 47.

Housewife.

ADMITTED: 7:3:23.

PRESENT ILLNESS: Duration: 3 months.  
Symptoms: Thirst and polyuria. Vulvitis.

PAST ILLNESSES: Appendix operation 1917.  
Prolapsus Uteri 1922.  
Menorrhagia 1923.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Skin dry. T. 101°: P. 120.  
R. 30. Oedema of ankles. Broncho Pneumonia.  
Vulva Red: Prolapsus Uteri. Urination  
painful. Urine- pus ++.

PROGRESS: Patient commenced treatment on a modified milk diet but later, with the clearing up of the urinary symptoms, the routine starvation dietary was instituted. On this regime sugar rapidly disappeared from the urine and did not recur with increasing diet. Patient left hospital much improved, free from glycosuria, on 50: 90: 120 = 1640.

	Admission.	Discharge.
Weight	88.2k.	83.8k.
Blood Sugar	0.18%	0.16%.
Urinary Sugar	55 gms.	Free.
Urinary Acetone	-	-

A case of mild diabetes with Broncho Pneumonia and chronic Cystitis.





Mrs. M.

Age 71.

Housewife.

ADMITTED: 27:3:23.

PRESENT ILLNESS: Duration: 6 months.  
 Symptoms: Attacks of giddiness since Oct. 1922. Thirst, frequency of micturition, polyuria since Jan. 1923. Sugar discovered Feb. 1923.

PAST ILLNESSES: Measles in childhood.  
 Scarlet Fever "  
 Whooping Cough "  
 Chicken-pox "  
 Erysipelas 1885.  
 Abscess of vulva 1921.

FAMILY HISTORY: Negative.

PRESENT CONDITION: Thin. Skin loose, glazed and pigmented. Considerable oedema of legs and feet. Arteries sclerosed. B.P. 150 mm. Hg. Lungs emphysematous. Tongue clean: constipated. Knee jerks not elicited: pupils normal.

PROGRESS: Patient shewed considerable acidosis on admission and treatment was commenced with oatmeal. In 3 days acetone had completely disappeared from the urine. The urine was sugar-free on oatmeal diet, but on increased feeding a trace of sugar appeared and persisted. The oedema gradually cleared up and the patient left hospital on 70: 70: 90 = 1370, much improved.

	Admission.	Discharge.
Weight	59.5k.	57.2k.
Blood Sugar	0.12%	0.15%.
Urinary Sugar	Nil.	Trace.
Urinary Acetone	+++	-

A case of glycosuria in a woman suffering from senility.





Ida Ross

Age 28

Hospital Nurse.

ADMITTED: 30:3:23.

PRESENT ILLNESS: Duration: 2½ years.

Symptoms: Polyuria since testing her own urine 2½ years ago and finding sugar present. Intermittent glycosuria since in spite of diet.

PAST ILLNESSES: Pneumonia in childhood.

Measles	"	"
Mumps	"	"
Scarlet fever	"	"
Diphtheria	"	"
Whooping Cough	"	"

FAMILY HISTORY: Negative.

PRESENT CONDITION: Well nourished, healthy looking girl. Skin moist: some scars right side of neck, due to old abscesses. Physical examination entirely negative.

PROGRESS: Glycosuria persisted in minimal amount quite irrespective of diet. The blood-sugar readings were always normal. A glucose test gave atypical "renal" result- a normal initial and final reading with a highest reading of 0.137g.

READMITTED: 23:1:24. Further tests confirmed the nature of the glycosuria. She was in excellent health and about to marry.



Name John Ross

Age 29

Ward 045 420  
Journal 040 410  
Page 035 400  
030 390  
025 380  
BLOOD 020 370  
SUGAR 015 360  
PER 010 350  
CENT 005 340

TOTAL  
URINARY  
SUGAR

Date of Admission...  
30-3-23

INSULIN UNITS.

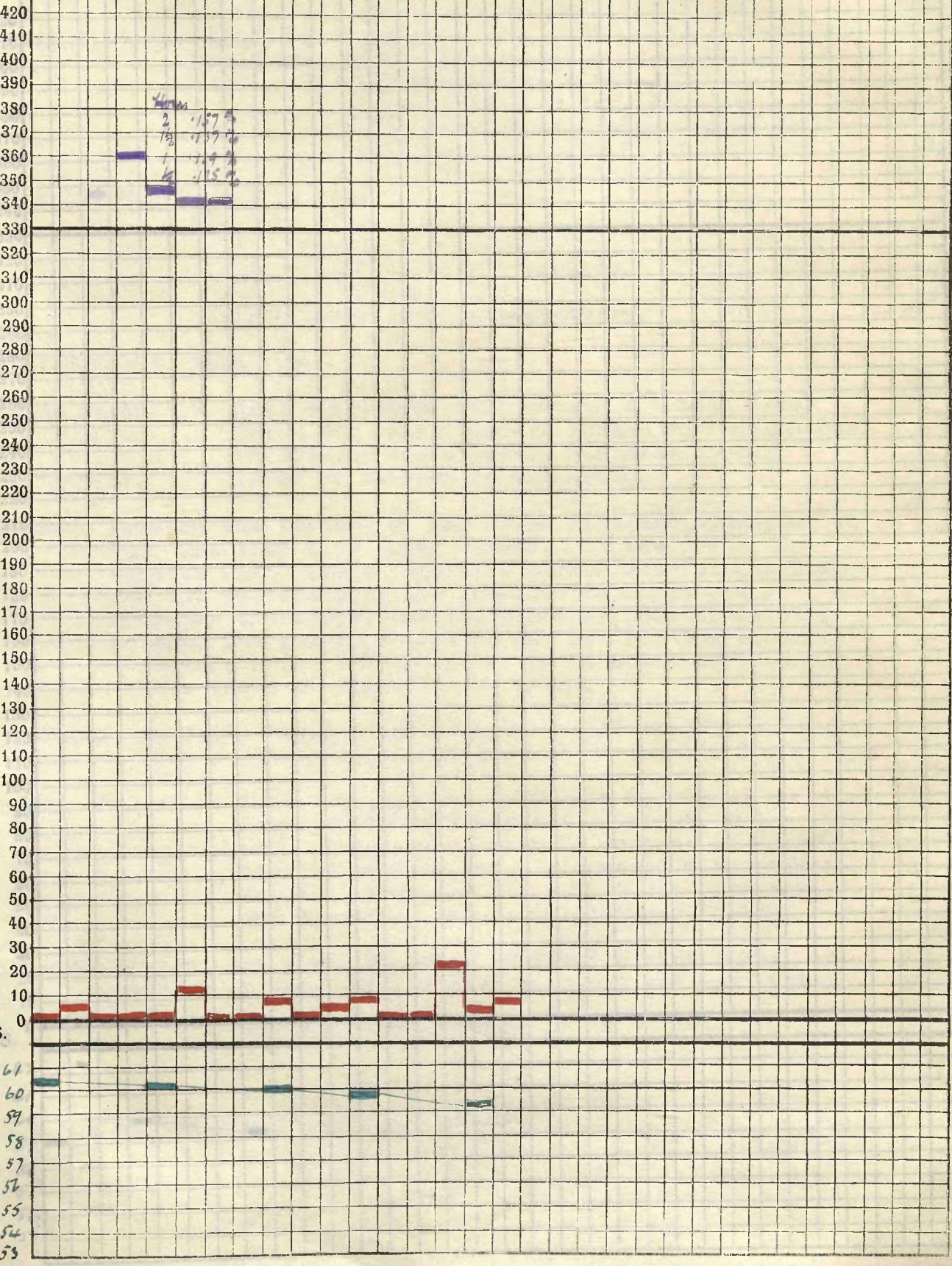
WEIGHT  
IN KILOS

DIET  
GRAMMES

C.	P.	F.	CALORIES
100	110	1790	"
"	"	"	"
"	"	"	"
60	25	385	"
30	10	160	"
"	"	"	"
40	30	720	"
"	"	"	"
45	70	865	"
"	60	1140	"
"	"	1320	"
"	"	"	"
200	100	2100	"
"	"	"	"
"	"	"	"
"	"	"	"

Date 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16

ACETONE - - - - - ++ + ++ ++ ++ + + + + + - - -





DIET		GRAMMES		F. CALORIES	
C.	P.	F.	C.	P.	F.
120	100	100	1780		
60	50	100	1340		
120	100	100	1780		

Name	Age	Weight	Journal	Page	Date	ACETONE
Ross	29 yrs.	6	54	164	23	-
					24	-
					25	-
					26	-
					27	+
					28	+
					29	+
					30	-
					31	-

BLOOD SUGAR PER CENT	0.45	0.40	0.35	0.30	0.25	0.20	0.15	0.10	0.05
420									
410									
400									
390									
380									
370									
360									
350									
340									
330									
320									
310									
300									
290									
280									
270									
260									
250									
240									
230									
220									
210									
200									
190									
180									
170									
160									
150									
140									
130									
120									
110									
100									
90									
80									
70									
60									
50									
40									
30									
20									
10									
0									

TOTAL URINARY SUGAR
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0

INSULIN UNITS.
62
61
60
59
58
57
56
55

WEIGHT IN KILOS
62
61
60
59
58
57
56
55



Name Mr McDonald Date 7

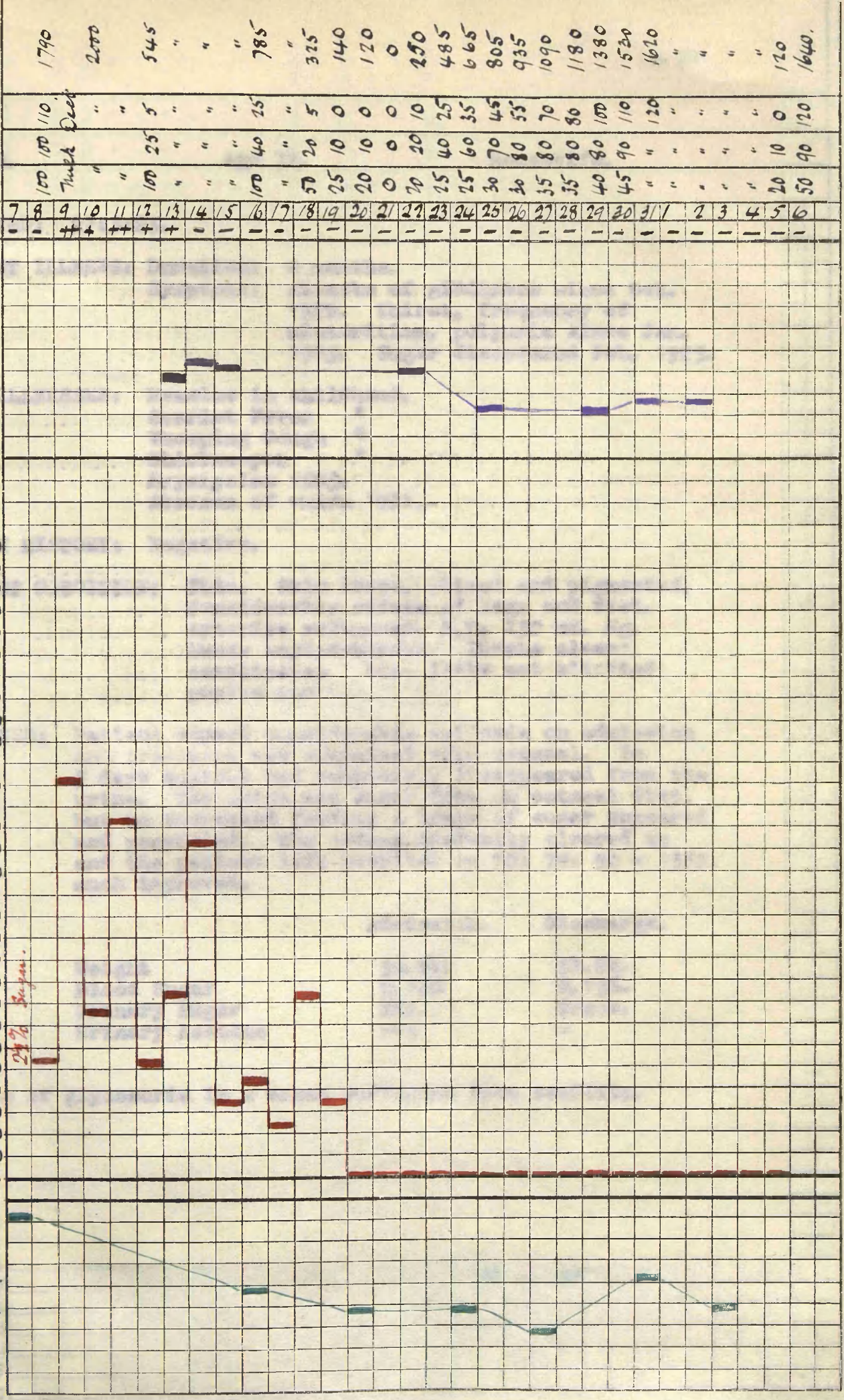
Age 47  
Ward .....  
Journal .....  
Page .....  
BLOOD SUGAR PER CENT  
0.45 420  
0.40 410  
0.35 400  
0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

TOTAL URINARY SUGAR

Date of Admission...  
7-3-13

INSULIN UNITS.

WEIGHT IN KILOS



Mrs. M.

Age 71.

Housewife.

ADMITTED: 27:3:23.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Attacks of giddiness since Oct. 1922. Thirst, frequency of micturition, polyuria since Jan. 1923. Sugar discovered Feb. 1923.

PAST ILLNESSES: Measles in childhood.  
Scarlet Fever "  
Whooping Cough "  
Chicken-pox "  
Erysipelas 1885.  
Abscess of vulva 1921.

FAMILY HISTORY: Negative.

PRESENT CONDITION: Thin. Skin loose, glazed and pigmented. Considerable oedema of legs and feet. Arteries sclerosed. B.P. 150 mm. Hg. Lungs emphysematous. Tongue clean: constipated. Knee jerks not elicited: pupils normal.

PROGRESS: Patient shewed considerable acidosis on admission and treatment was commenced with oatmeal. In 3 days acetone had completely disappeared from the urine. The urine was sugar-free on oatmeal diet, but on increased feeding a trace of sugar appeared and persisted. The oedema gradually cleared up and the patient left hospital on 70: 70: 90 = 1370, much improved.

	Admission.	Discharge.
Weight	59.8k.	57.2k.
Blood Sugar	0.12%	0.15%.
Urinary Sugar	Nil.	Trace.
Urinary Acetone	+++	-

A case of glycosuria in a woman suffering from senility.





Ida Ross

Age 28

Hospital Nurse.

ADMITTED: 30:3:23.

PRESENT ILLNESS: Duration: 2½ years.

Symptoms: ?Polyuria since testing her own urine 2½ years ago and finding sugar present. Intermittent glycosuria since in spite of diet.

PAST ILLNESSES: Pneumonia in childhood.

Measles " "

Mumps " "

Scarlet fever " "

Diphtheria " "

Whooping Cough " "

FAMILY HISTORY: Negative.

PRESENT CONDITION: Well nourished, healthy looking girl. Skin moist: some scars right side of neck, due to old abscesses. Physical examination entirely negative.

PROGRESS: Glycosuria persisted in minimal amount quite irrespective of diet. The blood-sugar readings were always normal. A glucose test gave atypical "Renal" result- a normal initial and final reading with a highest reading of 0.137g.

READMITTED: 23:1:24. Further tests confirmed the nature of the glycosuria. She was in excellent health and about to marry.



DIET		GRAMMES		F. CALORIES	
C.	P.	F.	C.	P.	F.
100	100	110	1790		
"	"	"	"		
"	"	"	"		
25	5	5	385		
10	0	0	160		
"	"	"	"		
50	30	30	730		
"	"	"	"		
70	45	45	865		
60	80	80	1140		
"	"	"	1320		
"	"	"	"		
100	100	100	2100		
"	"	"	"		
"	"	"	"		
"	"	"	"		

Name	Date	ACETONE
Mr. Rom	30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16	- - - - - ++ + ++ ++ ++ + + + + + - - -

Ward	Journal	Page	BLOOD SUGAR PER CENT
0.45	420		
0.40	410		
0.35	400		
0.30	390		
0.25	380		
0.20	370		
0.15	360		
0.10	350		
0.05	340		

TOTAL URINARY SUGAR
330
320
310
300
290
280
270
260
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0

INSULIN UNITS
61
60
59
58
57
56
55
54
53

WEIGHT IN KILOS
61
60
59
58
57
56
55
54
53







Mrs E.

Age 46

Housewife.

ADMITTED: 30:6:23.

PRESENT ILLNESS: Duration: 1 year.

Symptoms: Thirst, frequency of micturition,  
weakness, pruritis vulvae.Sugar discovered July 1922: dieted and  
well: stopped diet with return of  
symptoms.

PAST ILLNESSES: Cystitis 1901.

Pleurisy 1912.

" 1913.

Albuminuria 1913, with pregnancy.

" 1918.

Gastric Ulcer 1918: haematemesis.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished. Skin moist. V.V. both legs.  
Heart and lungs normal. Tongue furred: acetone  
in breath: constipated. Reflexes normal.  
Trace of albumen in the urine.PROGRESS: Patient was treated on the usual dietetic lines, but at  
1000 Cal. with C.70 sugar appeared in the urine. A  
Glucose Test gave a 'diabetic' curve. General nutrition  
was not satisfactory and insulin was commenced.  
Progress thereafter was satisfactory, patient gaining  
weight. The pulse ran around 80, but nothing abnormal  
discovered. She left hospital on 25 units of insulin,  
with a trace of sugar in the urine.READMITTED: 22:8:24. Patient had been well till 18:8:24 when she  
took a tonsillitis. She had had no insulin for 3 days.GENERAL CONDITION: Cold: comatose, and could not be roused. Pupils  
contracted: reflexes not elicited. Pulse 155,  
poor. Tongue very furred: acetone in breath:  
tonsils swollen, red, and covered with exudate.  
Urine: a trace of albumen. Sugar 2.5%. Acetone  
and Diacetic Acid ++. Blood Sugar = 0.40%.PROGRESS: Patient was given 20 units of insulin with 15gms. of  
Glucose at once. She revived somewhat and was able to  
talk, though rather confusedly.







1-42

DIET  
GRAMMES  
C. P. F. CALORIES

Diet To 12  
 Calmest  
 Water only

Name Mr. Gode Date 22 23

ACETONE + +

Weight 0.45 420  
 0.40 410  
 0.35 400  
 0.30 390  
 0.25 380  
 0.20 370  
 0.15 360  
 0.10 350  
 0.05 340  
 330  
 320  
 310  
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 220  
 210  
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 190  
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 120  
 110  
 100  
 90  
 80  
 70  
 60  
 50  
 40  
 30  
 20  
 10  
 0

BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

Date of Admission... 22/8/24

INSULIN UNITS. 40.60

WEIGHT IN KILOS

L. S. & Co. - 2000 - 4/24



Mrs B.

Age 66

Housewife.

ADMITTED: 12:9:23.

PRESENT ILLNESS: Duration: 1 year.

Symptoms: Thirst, polyuria, loss of weight, weakness,  
pain in back, pruritis vulvae.Sugar discovered Oct. 1922. Dieted with  
fair success. Sugar returned recently.PREVIOUS ILLNESSES: Tramway accident, followed by "rheumatics", June 1922.  
Cystitis July 1922.

Endometritis July 1922.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished: flabby. Tenderness over coccyx.  
Skin dry. Tongue furred: constipated. Reflexes  
normal.The urine contained Alb.+. Blood +. Pus +. B.Coli  
on culture.PROGRESS: Glycosuria was minimal on a reasonable diet (100:90:120=1340)  
throughout her residence. No insulin was required. A Glucose  
Test on 12:9:23 gave a frank Diabetic result. The B.Coli  
infection of the urinary tract was much improved.

A case of cystitis with glycosuria.

TOTAL  
URINARY  
SUGAR

0

10

20

30

40

50

60

70

80

90

100

110

120

130

140

150

160

170

180

190

200

210

220

230

240

250

260

270

280

290

300

310

320

330

340

350

360

370

380

390

400

410

420

430

440

450

460

470

480

490

500

510

520

530

540

550

560

570

580

590

600

610

620

630

640

650

660

670

680

690

700

710

720

730

740

750

760

770

780

790

800

810

820

830

840

850

860

870

880

890

900

910

920

930

940

950

960

970

980

990

1000







Rebecca Horner

Age 24

Clerkess.

ADMITTED: 18:9:23.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, frequency of micturition, loss of weight, weakness, constipation, swelling of feet.

Sugar discovered Dec. 1922. Dieted with little success.

PAST ILLNESSES: Measles in childhood.

Whooping-cough "

FAMILY HISTORY: Negative.

GENERAL CONDITION: Emaciated. Skin moist. Hair thin. Some oedema of ankles. Heart and lungs normal. Tongue coated: breath smelling of acetone; abdomen distended: constipated. Amenorrhoea for 4 months.

PROGRESS: Patient shewed considerable acidosis on admission, and treatment commenced with oatmeal diet. In spite of this acidosis persisted, and sugar was not reduced in the urine. Insulin was commenced on the 7th. day, and as oedema had become much more marked, the dose was rapidly increased to a maximum of 50 units with an increasing diet. Acetone quickly disappeared from the urine, but glycosuria continued and the B.S. ran constantly above normal limits despite numerous adjustments of diet and insulin dosage. Following 10 weeks of treatment the condition was much improved. On a diet of 60:80:110 = 1550 with 45-50 units of insulin only a trace of sugar was present in the urine. The oedema was never entirely absent throughout her residence.

	Admission	Dismissal
Weight	36.2k.	36.8k.
Blood Sugar	0.258%	0.202%
Urinary "	45gms.	Free
" Acetone	+++	-

A fairly satisfactory result in a case slow to respond to treatment.



Relief  
Home. Harv.

ge, ... 24

Ward ... 6

Journal . . . . .

age .....

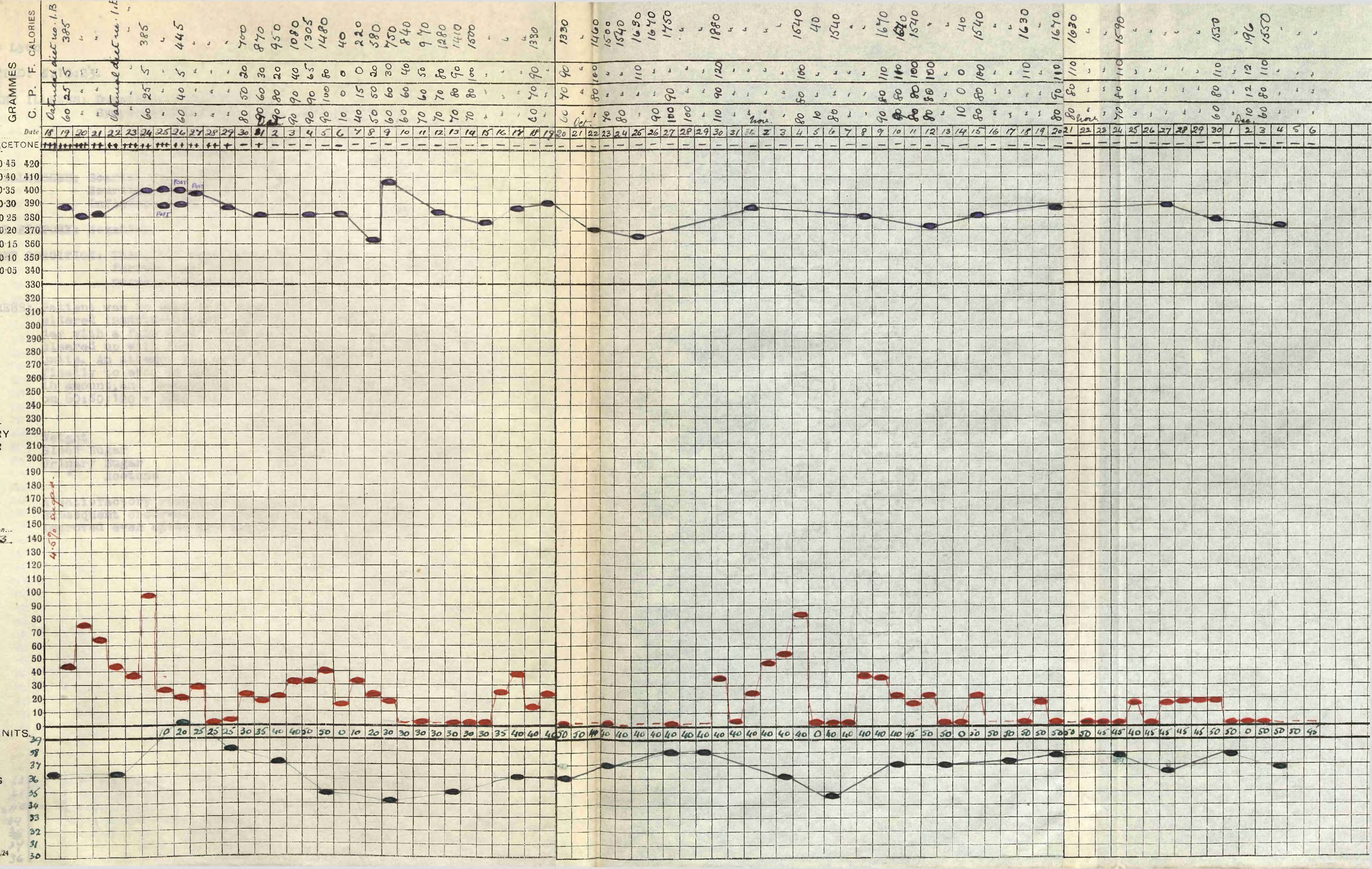
BLOOD  
SUGAR  
PER  
CENT

TOTAL  
URINARY  
SUGAR

Date of Admission...  
18. 9. 23

INSULIN UNITS

WEIGHT  
IN KILOS





Janey Lyon

Age 42

Nurse.

ADMITTED: 8:10:23.

PRESENT ILLNESS: Duration: 4 months.

Symptoms: Thirst, polyuria, frequency of micturition,  
loss of weight, weakness.Sugar discovered Aug. 1923. Dieted with no  
success.

PAST ILLNESSES: Scarlet fever 1892.

Neuritis following bicycle accident 1920.

Influenza 1922.

FAMILY HISTORY: Negative.

PRESENT CONDITION: Thin: ill nourished. Skin moist: no oedema. Tongue  
furred: considerable flatulence. Heart and lungs  
normal. Reflexes normal.

PROGRESS: Patient was so emaciated that starvation treatment was considered inadvisable, and insulin was commenced on the 2nd. day with a fair diet (60:40:20). Glycosuria was quickly cleared up with increasing doses of insulin, a maximum of 25 units. An attempt was then made to reduce the insulin and finally to stop it entirely: but sugar returned to the urine in amount, and insulin had to be resumed. Patient left hospital on 60:80:120 = 1640 with 10 units of insulin.

	Admission	Dismissal
Weight	41.5k.	41.8k.
Blood Sugar	0.45%	0.15%
Urinary Sugar	65gms.	Trace.
" Acetone	+	-

A satisfactory result in a moderately severe case. Subsequent observation shewed that the tolerance was not improved even after prolonged insulin treatment.







Margaret purves

Age 22

I.45  
Domestic-servant.

ADMITTED: 13:10:23.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Thirst, polyuria, frequency of micturition, loss of weight. Symptoms followed an attack of tonsillitis.

Sugar discovered 5 months ago.

PAST ILLNESSES: Chicken pox in childhood.

Influenza 1918.

Tonsillitis Jan. 1923.

" June 1923.

FAMILY HISTORY: Negative.

PRESENT CONDITION: Well nourished: healthy appearance. Skin moist. Some glossitis: breath has odour of acetone. Lungs-Expir. prolonged Rt. apex, with V. F. and V. R. +. Reflexes normal.

PROGRESS:

Patient shewed evidence of considerable acidosis on admission and commenced treatment on oatmeal diet. Acidosis persisting in spite of treatment insulin was administered and continued for a week in small dosage. Sugar and acetone rapidly disappeared, and patient made satisfactory progress thereafter on the usual dietetic lines. She left hospital on 65:95:130 = 1810, free from acetone and glycosuria.

TOTAL  
URINARY  
SUGAR

Weight

Blood Sugar

Urinary Sugar

" Acetone

Admission

59.8k.

0.25%

55gms.

+++

Dismissal

58.6k.

0.12%

Free

-

A satisfactory result. This case illustrates the temporary use of insulin to clear up acidosis and so expedite treatment.







Mrs N.

Age 25

Housewife.

ADMITTED: 15:10:23.

PRESENT ILLNESS: Symptoms: None. Sugar discovered on routine examination while in gynaecological wards for curettage. No glycosuria Dec. 1922 - on examination for insurance.

PAST ILLNESSES: Whooping cough in childhood.

Measles

"

Scarlet fever

"

FAMILY HISTORY: Negative.

GENERAL CONDITION: Physical examination entirely negative.

PROGRESS: A trace of sugar was constantly present in the urine, unaltered by the carbohydrate content of the diet. Three Glucose Tests gave results typical of Renal Glycosuria.

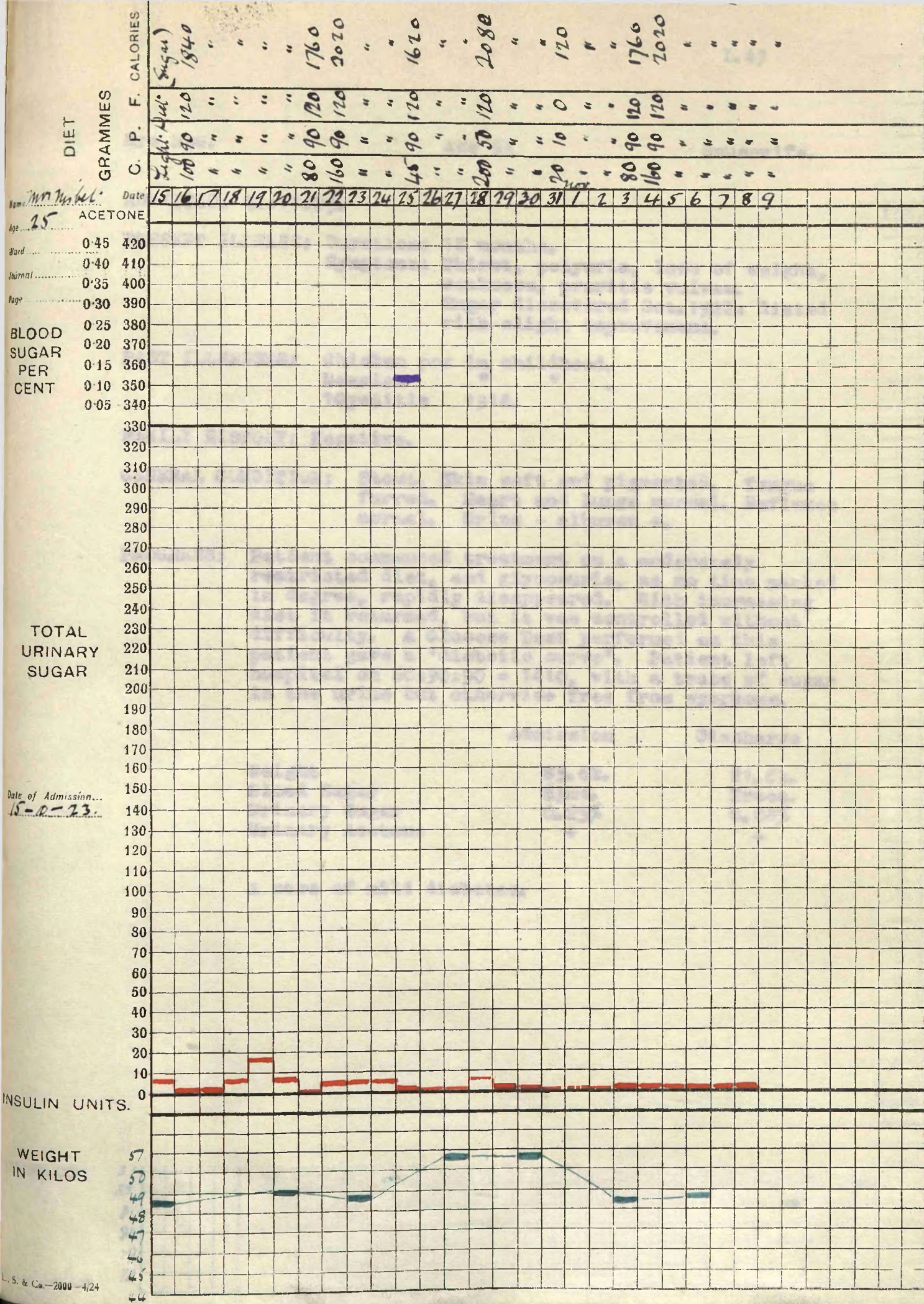
A case of Renal Glycosuria. No treatment required.

TOTAL  
URINARY  
SUGAR

330  
320  
310  
300  
290  
280  
270  
260  
250  
240  
230  
220  
210  
200  
190  
180  
170  
160  
150  
140  
130  
120  
110  
100  
90  
80  
70  
60  
50  
40  
30  
20  
10  
0

GRAMS UNITS







Mrs McG.

Age 55

Housewife.

ADMITTED: 02:11:23.

PRESENT ILLNESS: Duration: 18 months.

Symptoms: Thirst, polyuria, loss of weight, weakness, pruritis vulvae.

Sugar discovered Oct. 1922: dieted with slight improvement.

PAST ILLNESSES: Chicken pox in childhood.

Measles " " "

?Cystitis 1918.

FAMILY HISTORY: Negative.

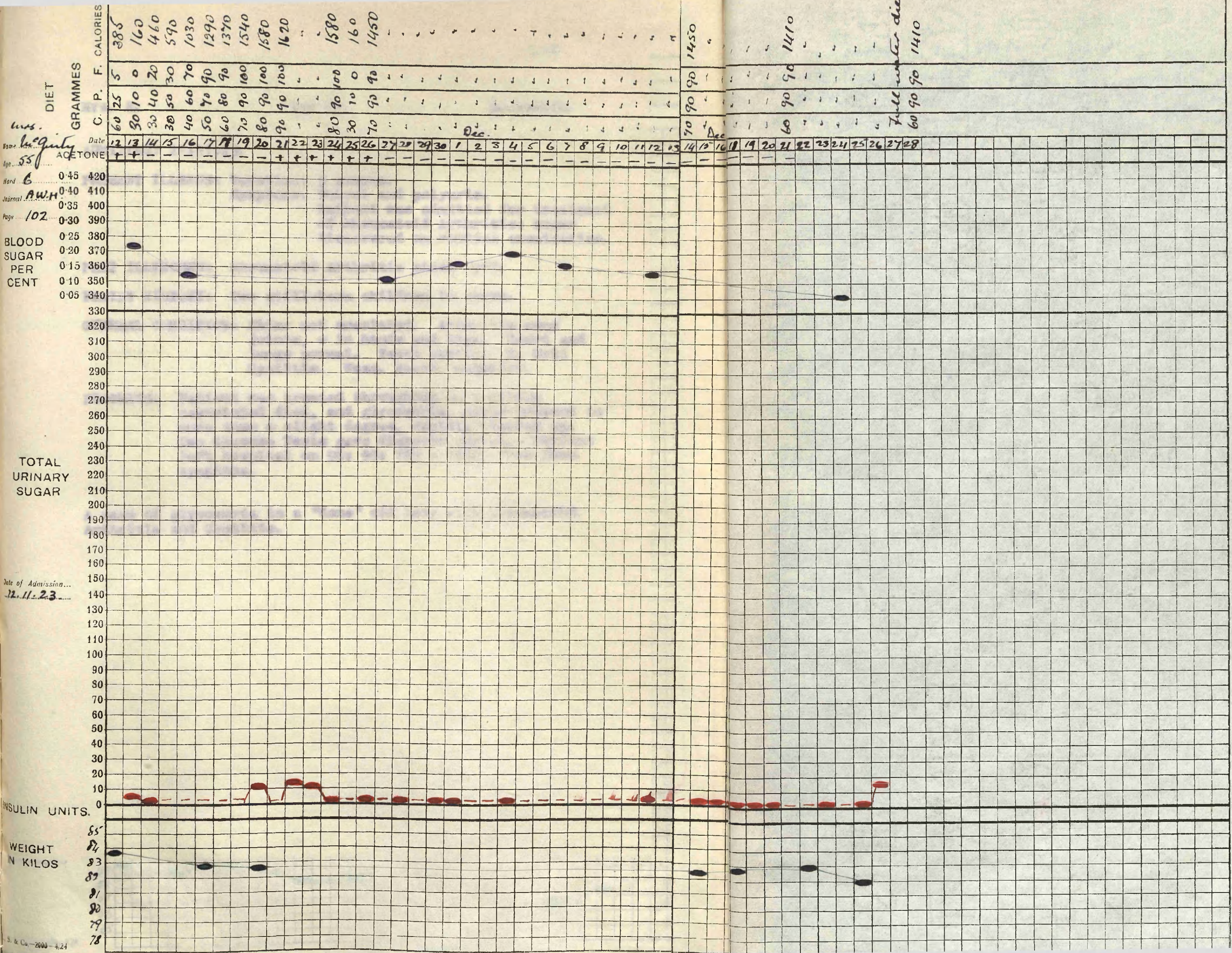
GENERAL CONDITION: Stout. Skin soft and pigmented. Tongue furred. Heart and lungs normal. Reflexes normal. Urine - albumen +.

PROGRESS: Patient commenced treatment on a moderately restricted diet, and glycosuria, at no time marked in degree, rapidly disappeared. With increasing diet it returned, but it was controlled without difficulty. A Glucose Test performed on this patient gave a 'diabetic curve'. Patient left hospital on 60:90:90 = 1410, with a trace of sugar in the urine but otherwise free from symptoms.

	Admission	Discharge
Weight	83.6k.	81.8k.
Blood Sugar	8gms.	Trace.
Urinary Sugar	0.25%	0.08%
Urinary Acetone	+	-

A case of mild diabetes.







Mrs. R.

Age 64.

Housewife.

ADMITTED: 21:11:23.

PRESENT ILLNESS: Duration: 3 months.

Symptoms: Thirst and polyuria.

Patient was admitted for treatment  
of Rheumatoid Arthritis: sugar  
discovered on routine examination.

PAST ILLNESSES: Rheumatoid Arthritis since 1917.

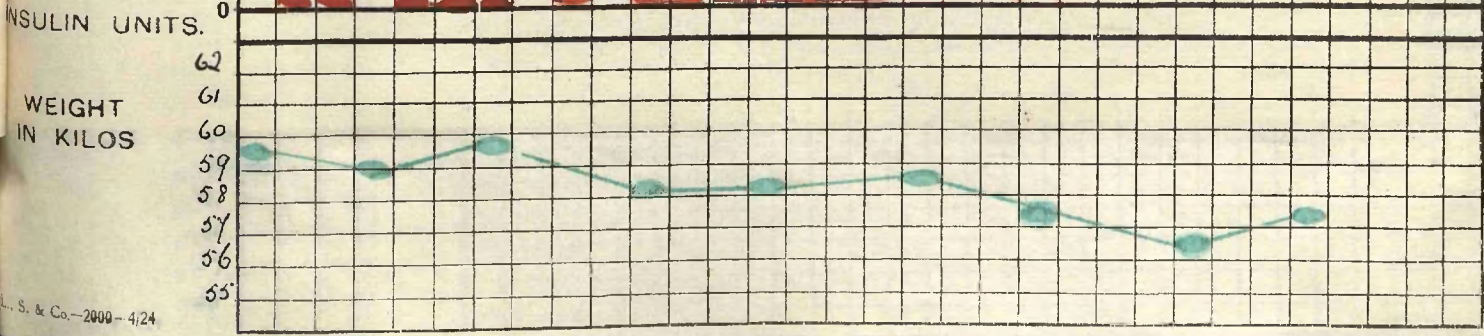
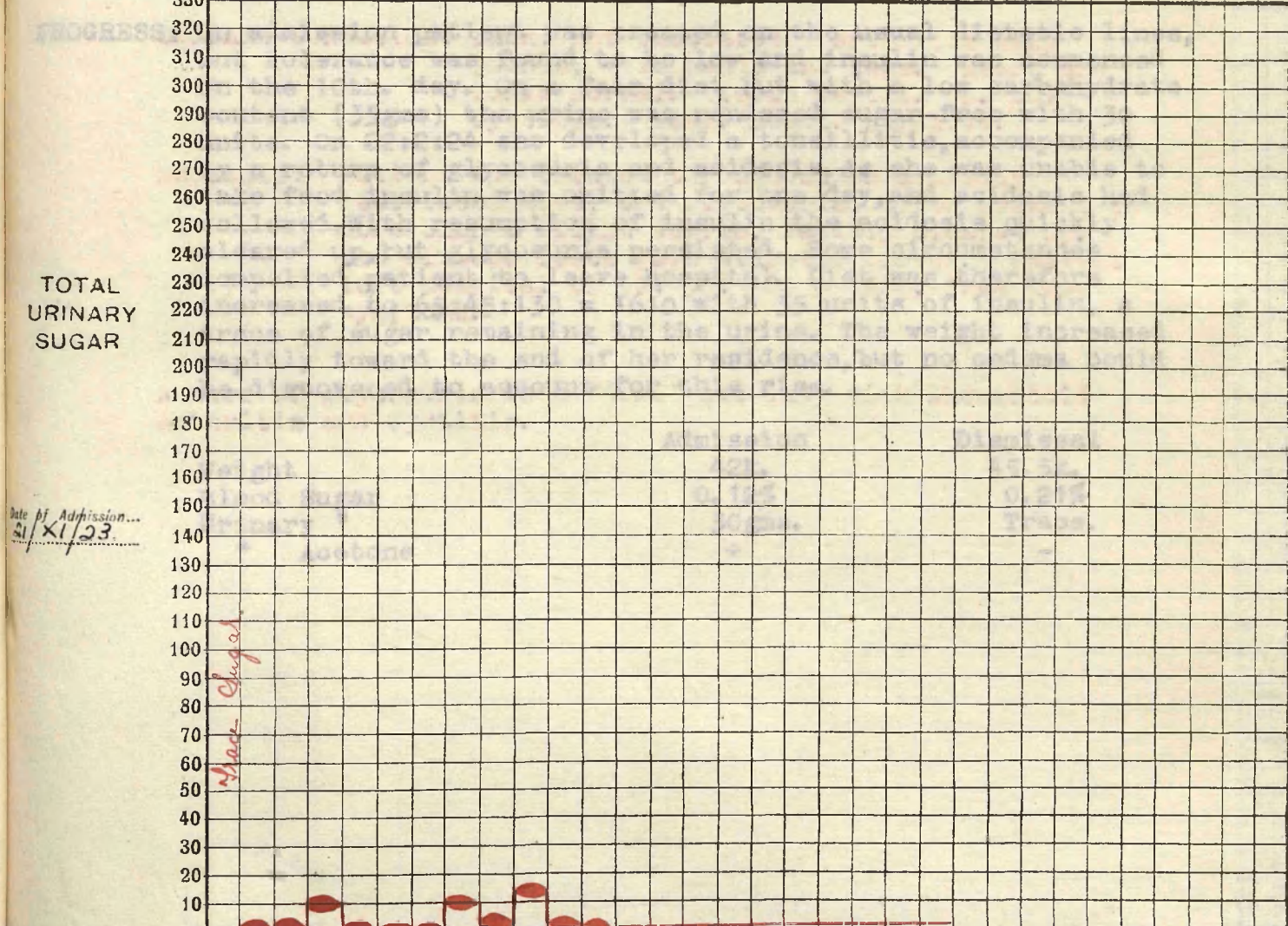
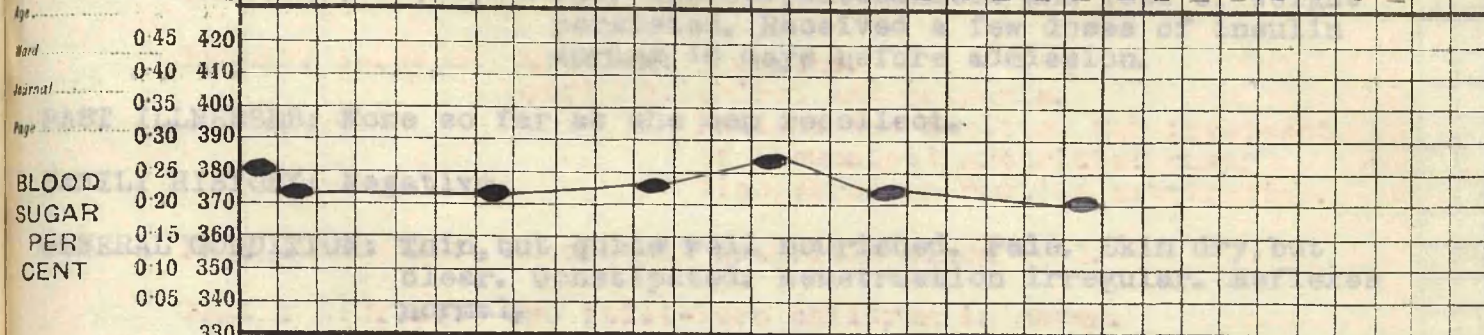
FAMILY HISTORY: Two still-born children in seven.

GENERAL CONDITION: Thin: not emaciated. Arthritis many  
joints, + in hands and legs. Heart and  
lungs normal. Teeth septic. B. Coli  
Cystitis. Wass. React. negative.PROGRESS: Patient was treated throughout on a mildly  
restricted diet, and glycosuria, never present to  
more than a slight degree, rapidly cleared up.  
Two Glucose Tests gave diabetic curves. Patient  
left hospital on 90: 90: 120 = 1500, free from  
symptoms.A case of glycosuria in a "done" old lady with Rheumatoid  
Arthritis and Cystitis.



DIET	GRAMMES	C.	P.	F.	CALORIES																								
						385.	1790.	1800																					
						5.	110	120																					
						25.	100	90																					

Name Robinson Date 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20





Lizzie Clark

Age 27

I.49  
Mill-worker.

ADMITTED: 13:12:23.

PRESENT ILLNESS: Duration: 9 months.

Symptoms: Thirst, polyuria, loss of weight, weakness, anorexia.

Sugar discovered 9 months ago: dieted with fair success, but weakness and loss of weight persisted. Received a few doses of insulin during 10 days before admission.

PAST ILLNESSES: None so far as she can recollect.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin, but quite well nourished. Pale. Skin dry, but clear. Constipated. Menstruation irregular. Reflexes normal.

PROGRESS: On admission patient was treated on the usual dietetic lines, but tolerance was found to be low and insulin was commenced on the 10th. day. On a fair diet but with a low carbohydrate content (35gms) the urine was rendered sugar-free with 30 units. On 22:2:24 she developed a tonsillitis, accompanied by a return of glycosuria and acidosis. As she was unable to take food insulin was omitted for one day, and acidosis had followed. With resumption of insulin the acidosis quickly cleared up, but glycosuria persisted. Home circumstances compelled patient to leave hospital. Diet was therefore increased to 65:45:130 = 1610 with 35 units of insulin, a trace of sugar remaining in the urine. The weight increased rapidly toward the end of her residence, but no oedema could be discovered to account for this rise.

	Admission	Dismissal
Weight	42k.	45.5k.
Blood Sugar	0.12%	0.21%
Urinary "	30gms.	Trace.
" Acetone	+	-



[illegible]



Mrs S. *Smith*

Age 59

Housewife.

ADMITTED: 22:12:23.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Thirst, polyuria, loss of weight, dimness of vision, pruritis vulvae, leucorrhoea.  
 Sugar discovered Sept. 1923. Dieted with little success.

PAST ILLNESSES: Measles in childhood.

FAMILY HISTORY: Negative.

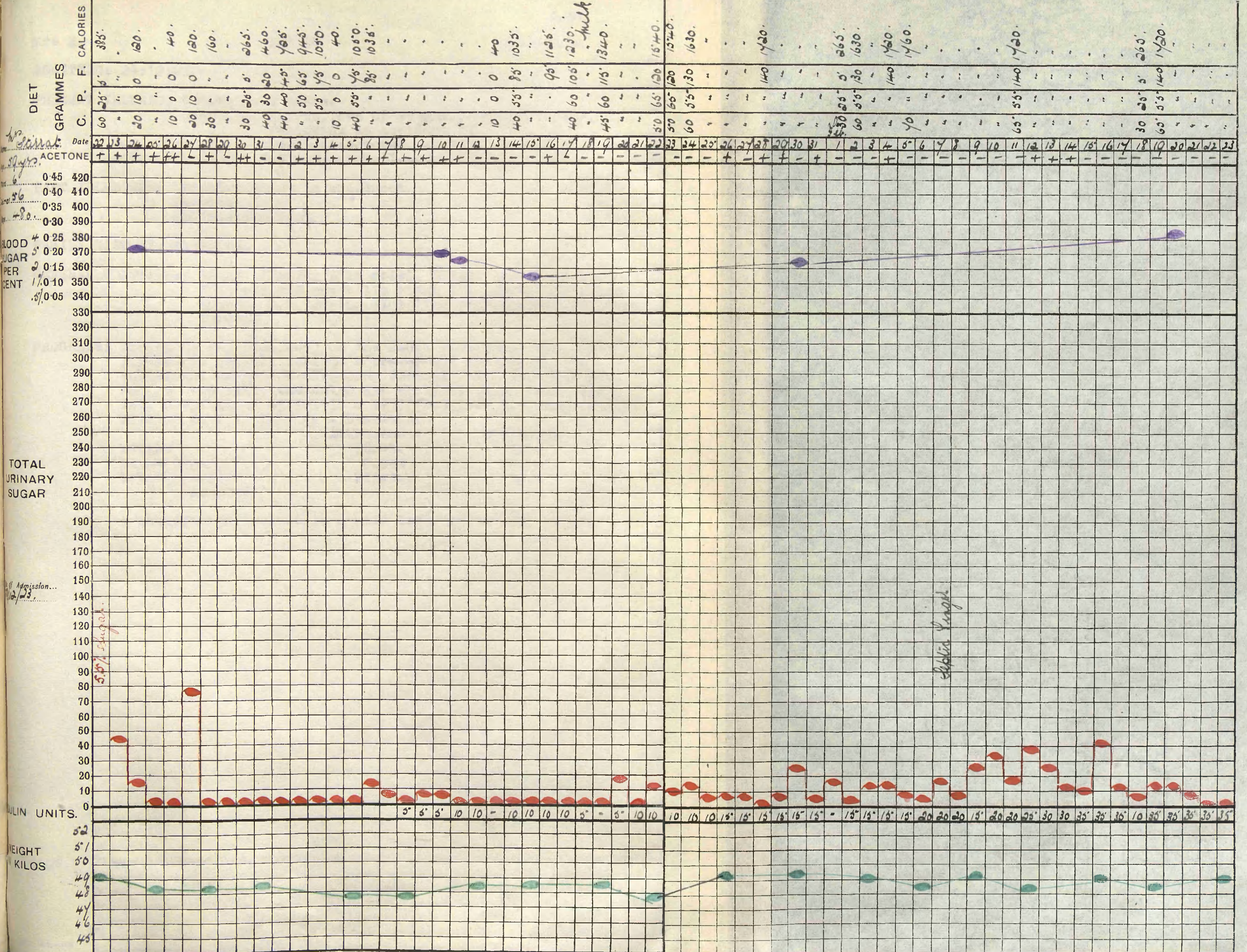
PRESENT CONDITION: Fairly stout. Skin dry and scaly: hair thin and falling out. Heart and lungs normal. Reflexes normal. Constipated. B.P. 110-70.

PROGRESS: Patient commenced treatment on the usual "Allen" lines, but it was found impossible to keep the urine sugar-free despite a fairly prolonged period of undernutrition dietary. Insulin was commenced 17 days after admission, and acetone and sugar were rapidly cleared from the urine. Some difficulty was experienced thereafter in keeping the urine sugar-free, and on 8:2:24, before this was accomplished, the patient developed an onychia which led to an exacerbation of the glycosuria. The dose of insulin was increased, but patient left hospital on 65:55:140 = 1720, with 35 units of insulin, and with a trace of sugar in the urine.

	Admission	Dismissal
Weight	49k.	49k.
Blood Sugar	0.21%	0.26%
Urinary Sugar	45gms.	Trace
" Acetone	+	-

A fairly satisfactory result in a refractory case. The influence of an intercurrent sepsis is clearly demonstrated.







Mrs H.

Age 58

Housewife.

ADMITTED: 24:12:23.

PRESENT ILLNESS: Duration: 7 years.

Symptoms: Thirst, loss of weight, pruritis vulvae,  
following an accident in 1916.Sugar discovered May 1917: dieted "off and  
on". Symptoms less marked recently.

PAST ILLNESSES: Whooping cough in childhood.

Measles "

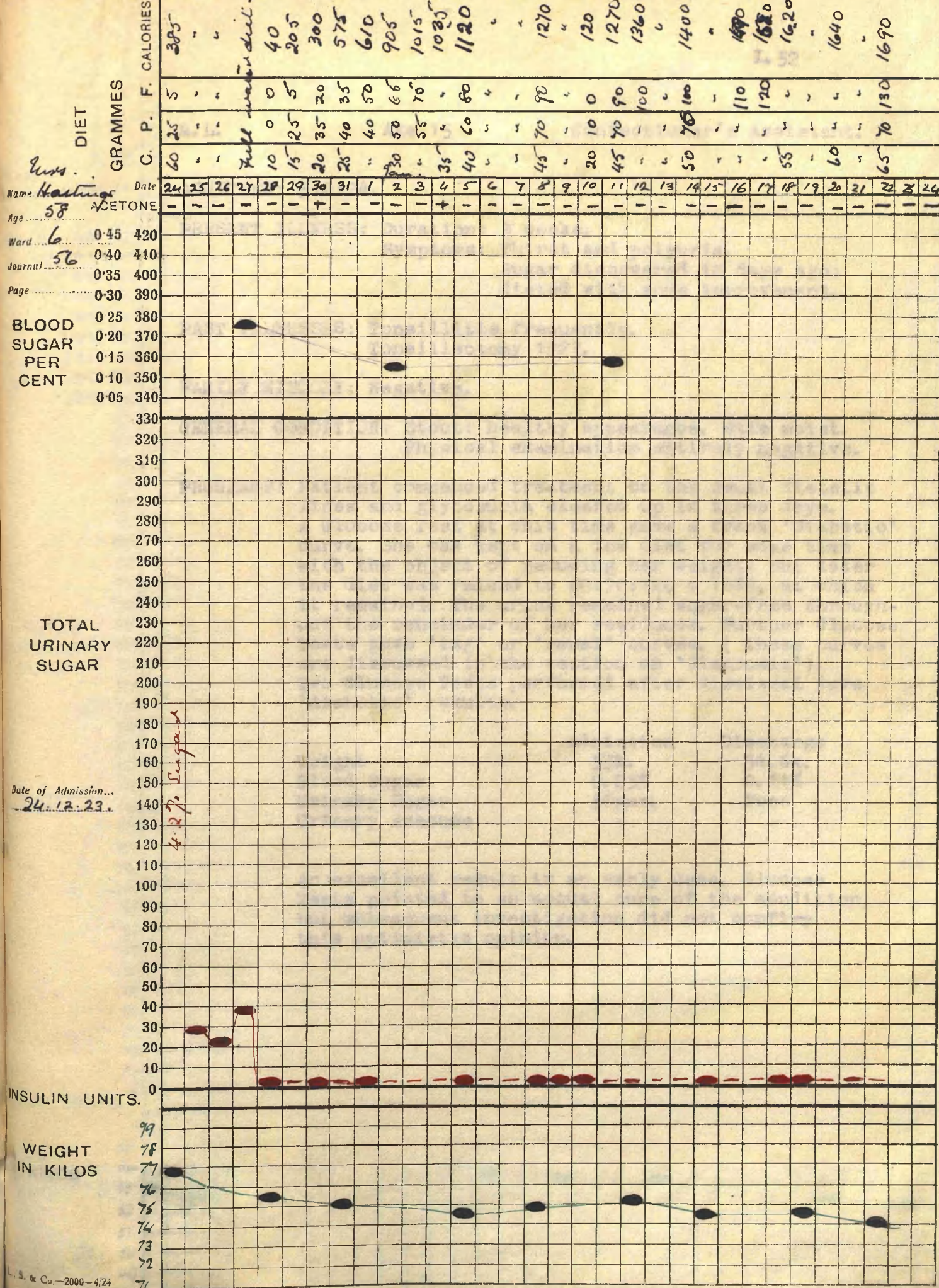
Indigestion 1895.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout: florid. Skin moist. Slight oedema of feet.  
Evidence old phlebitis right leg. Heart and lungs  
normal. B.P. 130-80. Glossitis: constipated. Reflexes  
normal.PROGRESS: Treatment was commenced on the usual dietetic lines. The  
glycosuria rapidly fell to a "trace," and throughout the  
subsequent period of advancing diet it never exceeded this  
amount. Generally the urine was entirely sugar-free. She  
left hospital on 65:70:130 = 1690, free from any symptoms.  
Glucose Test on 22:1:24 - Diabetic.

	Admission	Discharge
Weight	76.9k.	74k.
Blood Sugar	0.25%	0.14%
Urinary Sugar	28gms.	Free
" Acetone	-	-

A satisfactory result in a mild case. No insulin required.





M.L.

Age 15

Confectioner's Assistant.

ADMITTED: 5:2:24.

PRESENT ILLNESS: Duration: 3 weeks.

Symptoms: Thirst and polyuria.

Sugar discovered 10 days ago:  
dieted with some improvement.

PAST ILLNESSES: Tonsillitis frequently.

Tonsillectomy 1923.

FAMILY HISTORY: Negative.

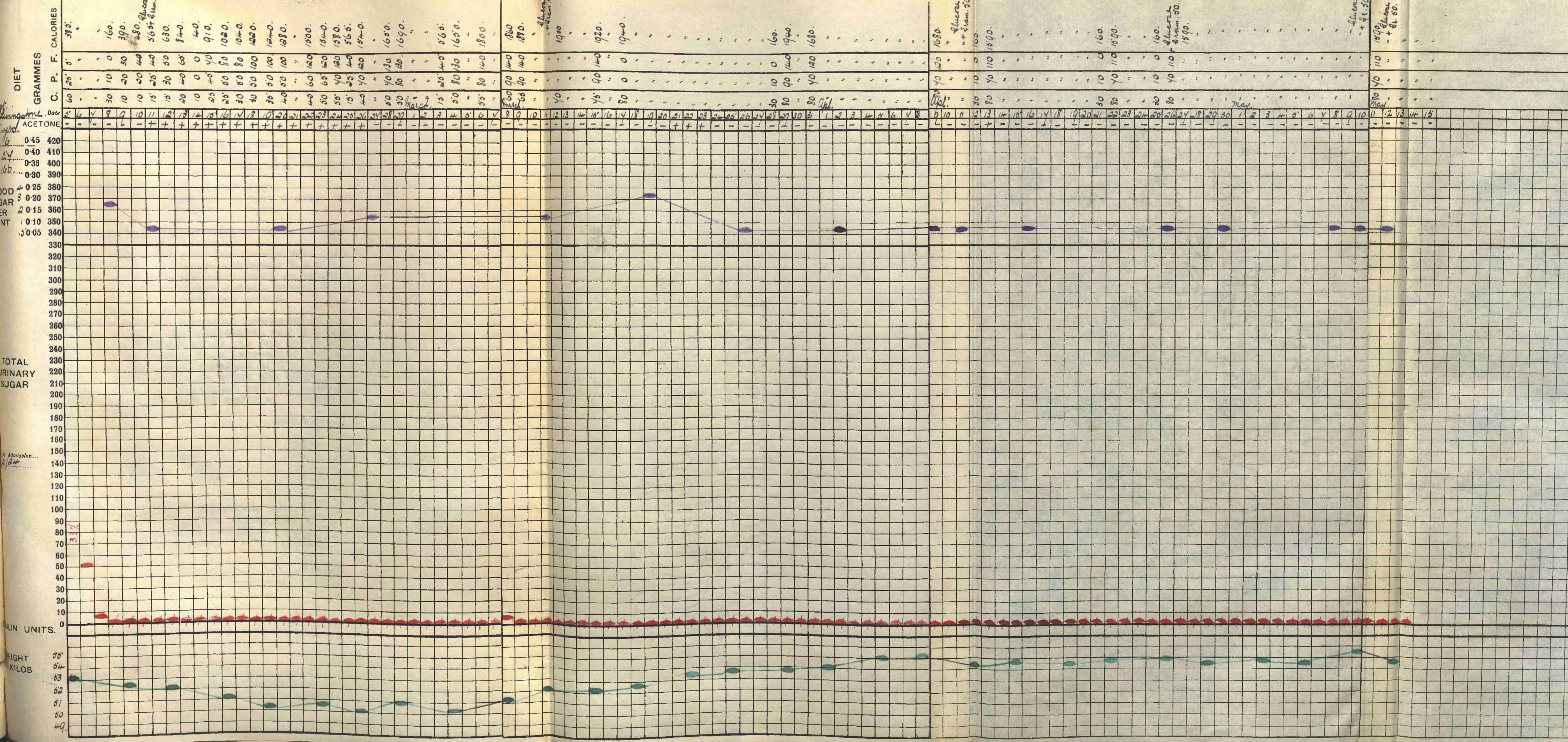
GENERAL CONDITION: Stout: healthy appearance. Skin moist.  
Physical examination entirely negative.

PROGRESS: Patient commenced treatment on the usual dietetic lines and glycosuria cleared up in three days. A Glucose Test at this time gave a frank 'Diabetic' curve. She was kept on a low diet for some time with the object of reducing her weight: but later the diet was raised to 80:70:120 = 1680, at which it remained. The urine remained sugar-free throughout the remainder of her residence. Further Glucose Tests gave 'lag' or 'renal' curves. ( These curves are discussed in the section on 'Diagnosis'). Two Glucose Tests performed after dismissal gave 'diabetic' results.

	Admission	Discharge
Weight	53k.	54.6k.
Blood Sugar	0.25%	0.11%
Urinary Sugar	50gms.	Free
Urinary Acetone	-	-

An excellent result in an early case. Glucose Tests pointed to an actual cure of the condition, but subsequent investigation did not confirm this optimistic opinion.



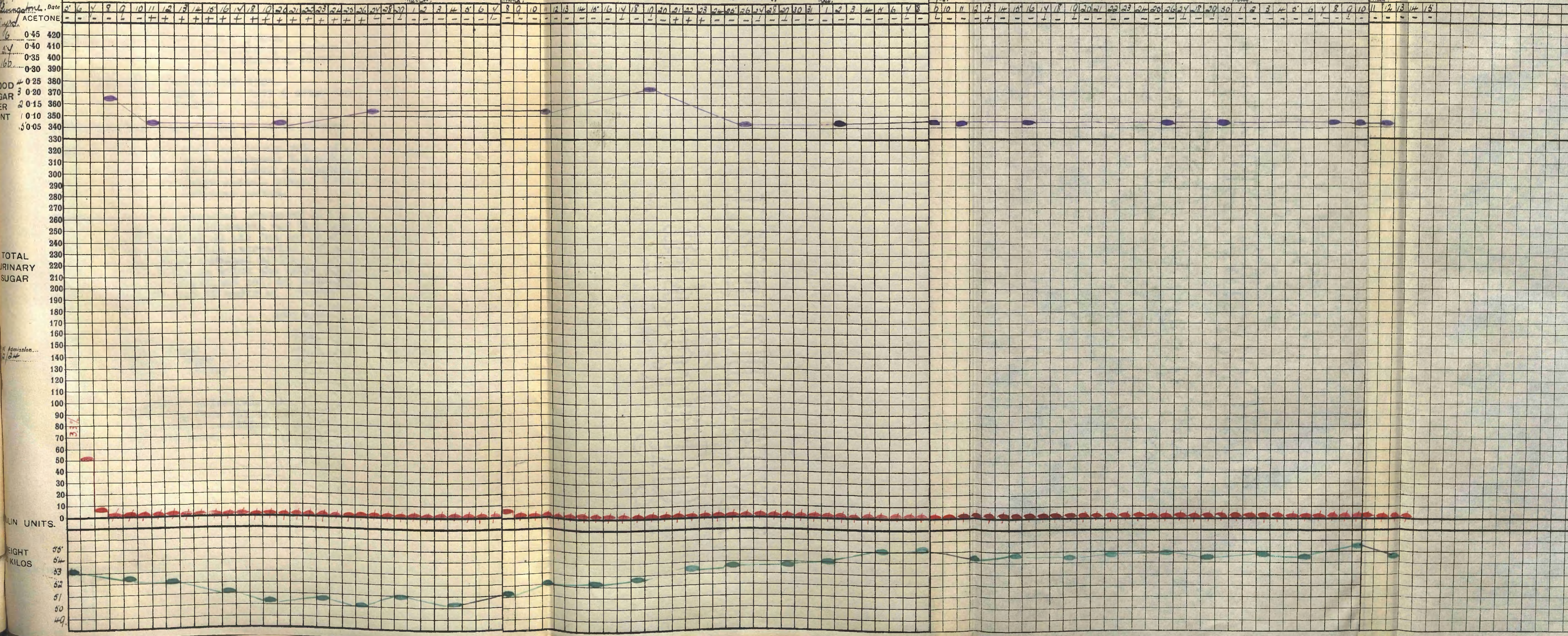


DIET	GRAMMES	P.	F.	CALORIES
60	25	8	38.5	
30	10	0	160.	
10	20	30	390.	
10	20	40	480. glucose	
15	25	45	565 + 4 am. 50	
15	30	50	630.	
20	40	65	840.	
10	0	0	40.	
25	45	70	910.	
25	60	80	1020.	
30	80	80	1040.	
30	100	100	1220.	
35	100	100	1240.	
40	100	100	1280.	
45	120		1500.	
50	120		1540.	
55	130		1580.	
55	140		1650.	
40	120		1540.	
50	130		1650.	
50	130		1650.	
55	140		1800.	

180	140	140	1800.
180	140	140	1800.
1900			glucose + 4 am. 50
1920.	90	140	
1940.	0	0	
	10	0	160.
	90	140	1940.
	40	120	1680.

1680.	140	140	1680.
glucose + 4 am. 50			
160.	10	0	160.
1690.	40	110	1690.
160.	10	0	160.
160.	40	110	1690.
160.	10	0	160.
glucose + 4 am. 50.	40	110	1690.
1590.			

1590.	140	140	1590.
glucose + 4 am. 50.			
1590.	40	110	1590.





Mrs L.

Age 37

Housewife.

ADMITTED: 10:2:24

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, loss of weight,  
weakness, breathlessness on exertion.

PAST ILLNESSES: 'Rheumatism' 1901.

Anaemia 1919 ( after last child ).

Measles 1921.

FAMILY HISTORY: Mother died of Heart Disease.

GENERAL CONDITION: Thin but fairly well developed. Skin moist.  
Heart enlarged: V.S.mitral murmur: 2nd.  
pulmonic sound +: numerous extrasystoles.  
Lungs normal. Tongue dirty: pyorrhoea.  
Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic lines, but glycosuria was obstinate and insulin was administered on the 8th. day on account of her poor condition. With increasing diet and insulin the glycosuria remained minimal, but it required 35 units of insulin to bring the urine sugar-free. The dose was later reduced, and patient left hospital on 80:65:130 = 1750 with 20 units of insulin.

	Admission	Discharge
Weight	41k.	44k.
Blood Sugar	0.12%	0.10%
Urinary Sugar	38gms.	Free
Urinary Acetone	+	-

A case of moderately severe diabetes in a woman suffering from mitral disease.  
The cardiac condition caused no discomfort throughout her residence.







Mrs M.

Age 56

Housewife.

ADMITTED: 1:4:24.

PRESENT ILLNESS: Duration: 5 months.

Symptoms: Thirst, polyuria, dimness of vision.  
 Sugar discovered 4 months ago:  
 dieted with improvement.

PAST ILLNESSES: Whooping Cough in childhood.

Measles " "

Operation for prolapse 1922.

Sclero-keratitis ~~1922~~ 1923.

FAMILY HISTORY: One brother died of Phthisis.

GENERAL CONDITION: Stout. Weak and 'flabby'. Skin moist: some  
 Psoriasis. Physical examination otherwise  
 negative.

PROGRESS: Patient commenced treatment on a low diet, but it  
 was considered unwise to starve her on account of  
 her poor condition. Insulin in small dosage was  
 therefore begun on the 6th. day with increasing diet.  
 Sugar and acetone rapidly disappeared from the urine.  
 Later insulin was reduced and finally stopped, the  
 diet remaining unchanged, and without return of  
 symptoms. She left hospital on 65:70:130 = 1710.

	Admission	Discharge
Weight	62.8k.	63.2k.
Blood Sugar	0.23%	0.09%
Urinary Sugar	74gms.	Free
Urinary Acetone	+	-

A satisfactory result in a mild case.







Mrs. G.

Age 42.

Housewife.

ADMITTED: 19:4:24.

PRESENT ILLNESS: Duration: Some years.

Symptoms: Weakness, pains in joints.

Sugar discovered December, 1923.

PAST ILLNESSES: Whooping Cough in childhood.

Measles " "

Prolapsus Uteri 1901.

Rheumatoid Arthritis 1914.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout: skin moist. Swelling and stiffness  
in many joints. Heart normal: B.P. 120  
mm. Hg. Lungs normal. Constipated.  
Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic  
lines. Sugar, present only in small amount on  
admission, rapidly disappeared and did not again  
reappear. Acidosis gave rise to no trouble.  
Patient left hospital free from glycosuria on  
60: 60: 130 - 1650. Insulin was never required.

Admission.

Discharge.

Weight

56k.

56.8k.

Blood Sugar

0.15%

0.10%.

Urinary Sugar

.5%

Free.

Urinary Acetone

-

-

A case of Rheumatoid Arthritis with glycosuria.  
A glucose test gave a diabetic curve.



GRAMMES

C. P. F. CALORIES

Name: Mr. Goodman Date: \_\_\_\_\_

Age, 42..... ACETONE

Word 6 0-45 420

Journal 0.4 0.40 410

0.35 400

Page ... 0.30 390

BLOOD # 0.25 380

SUGAR	370
-------	-----

PER 2% 0.15 360

CENT	1% 0-10	350
	1% 2-25	350

3% 0-05	340
---------	-----

TOTAL  
URINARY  
SUGAR

Date of Admission...

5/3/24

INSULIN UNITS.

WEIGHT  
IN KILOS

59  
58  
54  
56  
55  
54  
53

6% Engrs.



I.R.

Age 9

Schoolgirl.

ADMITTED: 19:5:24.

PRESENT ILLNESS: Duration: 3 weeks.

Symptoms: Thirst, polyuria, loss of weight,  
headache.

Sugar discovered 2 weeks before.

PAST ILLNESSES: Whooping Cough 1920.

Scarlet Fever 1920.

Measles 1920.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin and emaciated. Skin dry. Heart and  
lungs normal. Tongue coated: abdomen  
normal. Reflexes normal.

PROGRESS: Patient was treated on the usual dietetic lines, commencing with the second diet of the scale. Without actual starvation the sugar disappeared from the urine in 8 days, while with increasing diet acetonuria gradually cleared up. Thereafter save on rare occasions the urine remained sugar-free, but no increase in weight occurred until the diet reached 60-70 Calories per kilo body weight. She left hospital on 50:60:125 = 1565, free from symptoms.

	Admission	Discharge
Weight	22.7k.	23.4k.
Blood Sugar	0.21%	0.08%
Urinary Sugar	25gms.	Free.
Urinary Acetone	++	-

A case of moderately severe diabetes in a child. This case illustrates clearly the large food-intake necessary in the child to permit of normal growth.







A. T.

Age 31.

Baker's Shop Assistant.

ADMITTED: 14:7:24.

PRESENT ILLNESS: Duration: 5 months.

Symptoms: Thirst, polyuria, loss of weight, pruritis  
vulvae. Great craving for sweet cakes.  
Sugar discovered 2 months ago. Dieted with  
little result.

PAST ILLNESSES: Ruptured Varicose Vein 1918.

Metrorrhagia: Leucorrhoea 1922.

Curettage 1923.

FAMILY HISTORY: Father died of Phthisis.

GENERAL CONDITION: Well nourished: stout. Skin moist. No oedema.  
Heart and lungs normal. Reflexes normal. Tongue  
coated: odour of acetone in breath.

PROGRESS: Patient commenced treatment on the usual dietetic lines,  
but acidosis, considerable on admission, remained unchanged.  
Insulin was therefore administered on the 5th. day. The  
acetone and diacetic acid were rapidly cleared from the  
urine, but sugar persisted in small amount. ON 70:60:110 =  
1510 insulin 30 units were required to clear the glycosuria,  
but patient left hospital 3 weeks later on the same diet  
free from glycosuria on 18 units of insulin.

	Admission	Discharge
Weight	63.5k.	61k.
Blood Sugar	0.354%	0.08%
Urinary "	78gms.	Free
" Acetone	++	-

A satisfactory result in a case of moderate severity.







Mrs D.

Age 52

Housewife.

ADMITTED: 24:7:24.

PRESENT ILLNESS: Duration: 2½ years.

Symptoms: Thirst, polyuria, loss of weight,  
weakness, pruritis vulvae.  
Sugar discovered 8 weeks before.PAST ILLNESSES: Whooping Cough in childhood.  
Measles in childhood.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Skin dry. Varicose veins both legs.  
Heart normal: B.P. 150mm.Hg. Lungs normal.  
Reflexes normal. Tongue furred.PROGRESS: Glycosuria, never marked in degree, quickly cleared  
up restricted diet without resort to starvation.  
With increasing diet a trace of sugar returned to  
the urine, but this was readily controlled.  
Patient left hospital on 80:60:120 = 1640, free  
from symptoms.

## Admission

## Discharge

Weight

71.2k.

68k.

Blood Sugar

0.18%

0.10%

Urinary Sugar

17gms.

Free

Urinary Acetone

-

-

A case of mild diabetes.



Ward 6

Journal P.W.H.

Page 174

BLOOD SUGAR PER CENT

0.45 420

0.40 410

0.35 400

0.30 390

0.25 380

0.20 370

0.15 360

0.10 350

0.05 340

330

320

310

300

290

280

270

260

250

240

230

220

210

200

190

180

170

160

150

140

130

120

110

100

90

80

70

60

50

40

30

20

10

0

INSULIN UNITS.

WEIGHT IN KILOS

74

73

72

71

70

69

68

67

DIET		GRAMMES		C.		P.		F.		CALORIES																						
24	25	26	27	28	29	30	31	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	
ACETONE		-	-	+	-	-	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
BLOOD SUGAR PER CENT		0.18	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	
TOTAL URINARY SUGAR		0.25	0.20	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	0.15	
INSULIN UNITS		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
WEIGHT IN KILOS		71.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	70.5	



Mrs M.

Age 58

Housekeeper.

ADMITTED: 24:7:24.

PRESENT ILLNESS: Duration: 18 months.

Symptoms: Thirst, polyuria, frequency of micturition, loss of weight.

Patient also suffered from a cancer of the breast. Sugar was discovered when admitted for operation, and she was transferred for treatment.

PAST ILLNESSES: Measles in childhood.

Whooping Cough "

Scarlet Fever "

Smallpox 1878.

Typhoid 1881.

Rheumatic Fever 1911.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Scirrhus cancer Rt. breast. Skin moist.  
Slight oedema of feet. Heart and lungs normal.  
Tongue coated. Reflexes normal.

PROGRESS: Progress was uneventful. A very moderate restriction of Carbohydrate intake was sufficient to clear up the glycosuria. Patient left hospital on 100:75:100 = 1600 free from symptoms.

	Admission	Discharge
Weight	87.8k.	86k.
Blood Sugar	0.22%	0.10%
Urinary Sugar	18gms.	Free
" Acetone	-	-

A satisfactory result in a mild case.

Sept. 1924. Patient was operated upon for removal of Rt. breast. The operation passed off without incident, and she made an uninterrupted recovery.







Mrs R.

Age 58

Housewife.

ADMITTED: 13:8:24.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Pruritis vulvae 1921: dieted with success  
Taking ordinary diet of late. Thirst,  
polyuria, and later pains in epigastrium,  
vomiting, and drowsiness.

PREVIOUS ILLNESSES: None so far as she can recollect.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Fairly well nourished. Appears to have lost flesh  
Skin dry. Heart not enlarged: apical V.S.murmur.  
Pulse 100. Some rale both bases. Tongue furred:  
pyorrhoea. Reflexes normal. Urine contains a  
trace of albumen. Odour of acetone in the breath.  
Drowsy and aroused with difficulty.

PROGRESS: On admission patient was apparently on the verge of coma,  
and treatment commenced with oatmeal and insulin. 50  
units were administered in the first 24 hours, with in  
addition, 15gms. of glucose. Her condition improved very  
rapidly, and insulin was stopped for the following 3 days.  
On account of persistent glycosuria it was begun again,  
and increased to 30 units when the urine became sugar-  
free. Thereafter insulin was gradually reduced in amount  
with a constant diet until finally it was entirely omitted  
without return of glycosuria. Save for slight oedema of  
the feet she left hospital free from symptoms on 75:85:120  
= 1810.

	Admission	Discharge
Weight	60.5k.	59.5k.
Blood Sugar	0.296%	0.14%
Urinary "	25gms.	Free
" Acetone	++	-

A very satisfactory result in an apparently severe case.  
Carbohydrate tolerance would appear to have been improved  
by a period of insulin treatment.







Mrs McK.

Age 53.

Housewife.

ADMITTED: 12:9:24.

PRESENT ILLNESS: Duration: 2 years.

SYMPTOMS: Thirst, polyuria, frequency of micturition,  
loss of weight, weakness, pruritis vulvae.

PAST ILLNESSES: None.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Fat. Skin healthy. Varicose veins both legs.  
Heart and lungs normal. Tongue clean. Reflexes  
normal.PROGRESS: Progress was uneventful. Glycosuria was rapidly dispelled  
by diet alone. With increasing diet a trace of sugar was  
occasionally present in the urine, easily cleared up by a  
day's starvation. Acidosis was never troublesome. She  
left hospital on 65:70:130 = 1710, free from symptoms.

A satisfactory result in a mild case.

TOTAL  
URINARY  
SUGAR

UNITS



No. *W. King*  
 Name *W. King*  
 Age *53*  
 Ward *6*  
 Journal *29*  
 Page *124*

BLOOD SUGAR PER CENT  
 0.45  
 0.40  
 0.35  
 0.30  
 0.25  
 0.20  
 0.15  
 0.10  
 0.05

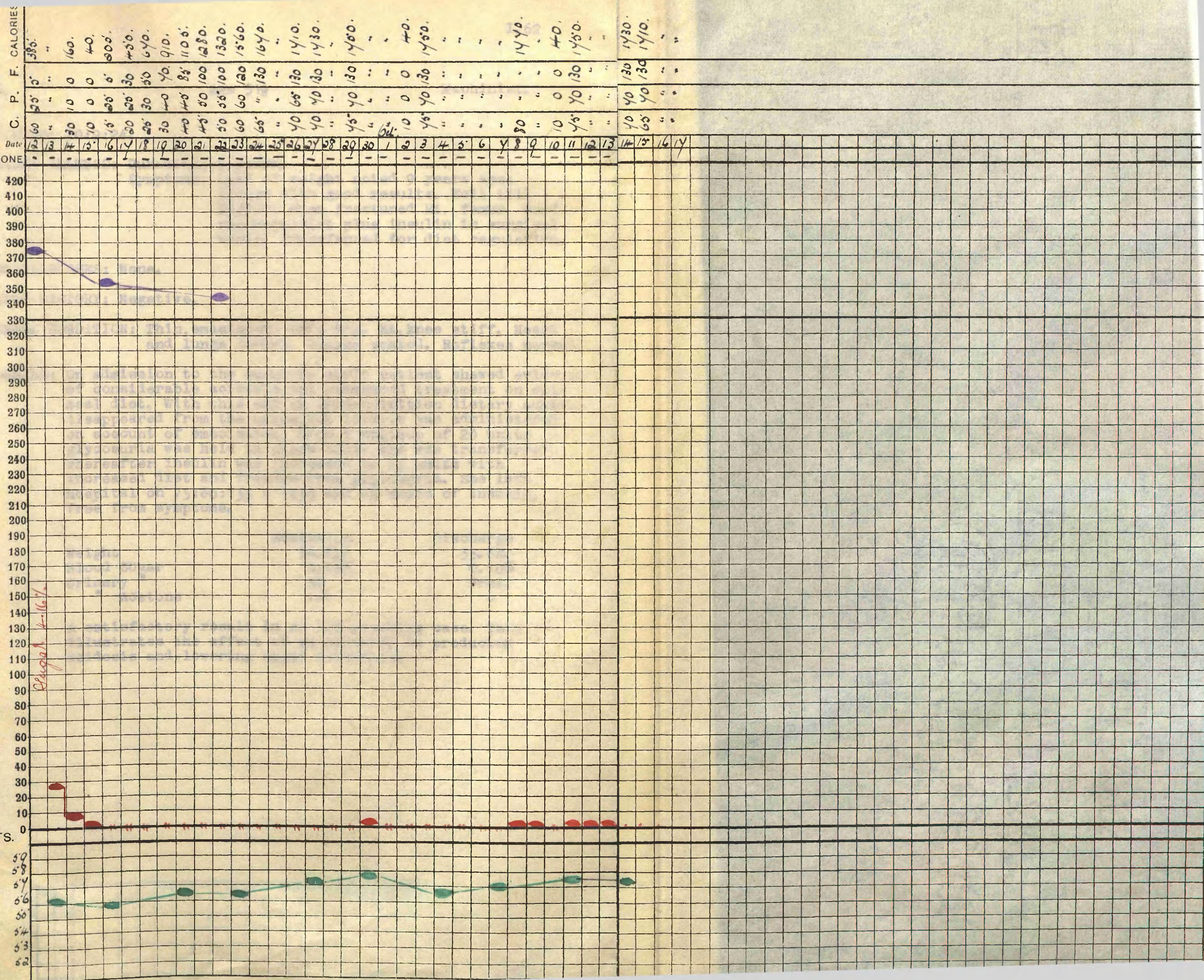
TOTAL URINARY SUGAR

Date of Admission...  
*12/8/24*

INSULIN UNITS.

WEIGHT IN KILOS

DIET GRAMMES





C.B.

Age 51½

Machinist.

ADMITTED: 20:9:24.

PRESENT ILLNESS: Duration: 9 years.

Symptoms: Loss of weight noted 9 years ago:  
 dieted with good results. Well till  
 24:7:24 when fractured Rt. femur. Good  
 recovery: diet plus insulin in surgical  
 wards. Transferred for diet regulation.

PAST ILLNESSES: None.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin, emaciated. Skin dry. Rt. knee stiff. Heart  
 and lungs normal. Tongue coated. Reflexes normal.

PROGRESS: On admission to the surgical wards patient shewed evidence  
 of considerable acidosis, and commenced treatment on oat-  
 meal diet. With this and an undernutrition dietary acetone  
 disappeared from the urine, but insulin was administered  
 on account of emaciation. With a maximum of 20 units  
 glycosuria was held in check until she was transferred.  
 Thereafter insulin was increased to 25 units with  
 increased diet and freedom from glycosuria. She left  
 hospital on 75:80:135 = 1835 and 25 units of insulin,  
 free from symptoms.

	Admission	Discharge
Weight	34.8k.	35.8k.
Blood Sugar	0.24%	0.10%
Urinary "	5%	Free.
" Acetone	+++	-

A satisfactory result in an old-standing case. This case  
 illustrates the effect of an accident in producing  
 acidosis and lowering sugar tolerance.







Mrs G.

Age 42

Housewife.

ADMITTED: 11:11:24

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Polyuria, pain in back, occasional oedema of feet.

Sugar discovered June 1924 : dieted with some improvement.

PAST ILLNESSES: Scarlet Fever 1898.

Acute Rheumatism 1898.

Appendicectomy 1921 : Peritonitis.

FAMILY HISTORY: Good.

GENERAL CONDITION: Very stout. Skin moist. Organs apparently normal, but examination difficult on account of fat. Teeth septic. Reflexes normal.

PROGRESS: Patient commenced treatment upon a moderately restricted diet and glycosuria rapidly disappeared. Even with 50 gms. of Glucose added to the diet for test purposes the urinary sugar was merely a trace. The Glucose Test gave a Diabetic curve. Patient left hospital irregularly after 5 days, declining to continue the treatment.

	Admission	Discharge
Weight	95.8k.	94.2k.
Blood Sugar	Trace	Free
Urinary Sugar	0.15%	-
Urinary Acetone	-	-

A case of mild Diabetes in a woman suffering from obesity.



*Mr. Gundrod*

Name *Gundrod* Date *11/11/24*

Age *6* ACETONE

Ward *6* 0.45 420

Journal *59* 0.40 410

Page 0.35 400

0.30 390

BLOOD 0.25 380

SUGAR 0.20 370

PER 0.15 360

CENT 0.10 350

0.05 340

330

320

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INSULIN UNITS.

98

97

96

95

94

93

92

91

WEIGHT  
IN KILOS

98

97

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Mary McI.

Age 57

Dressmaker.

ADMITTED: 27:10:24.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Polyuria, frequency of micturition,  
weakness, dimness of vision.PAST ILLNESSES: Similar symptoms 15 years ago: sugar discovered  
in the urine: dieted: symptoms disappeared in a  
short time and did not recur.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Small: thin: rhachitic. Skin moist. Hair thin.  
Heart and lungs normal. Constipated. Reflexes  
normal. Vision very defective. Urine contains  
albumen ++, pus and a few casts.PROGRESS: Patient commenced treatment on the usual dietetic lines.  
Glycosuria was minimal, and quickly cleared up. The  
general condition, however, was so unsatisfactory that  
insulin was administered on the 13th. day, and increased  
to 15 units. She left hospital on 80:60:130 = 1730 with  
10 units of insulin, free from symptoms.

	Admission	Discharge
Weight	44.2k.	45.7k.
Blood Sugar	0.27%	0.22%
Urinary "	Trace	Free
" Acetone	-	-

A satisfactory result in a case of interstitial nephritis  
and cystitis, and mild diabetes.



Mary  
Name: *M. M. M.* Date: *27/28/29/30/31/1/2/3/4/5/6/7/8/9/10/11/12/13/14/15/16/17/18/19/20/21/22/23/24/25/26/27*

Age: *54*  
Ward: *6*  
Journal: *59*  
Page: *6*  
BLOOD SUGAR PER CENT  
0.45 420  
0.40 410  
0.35 400  
0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

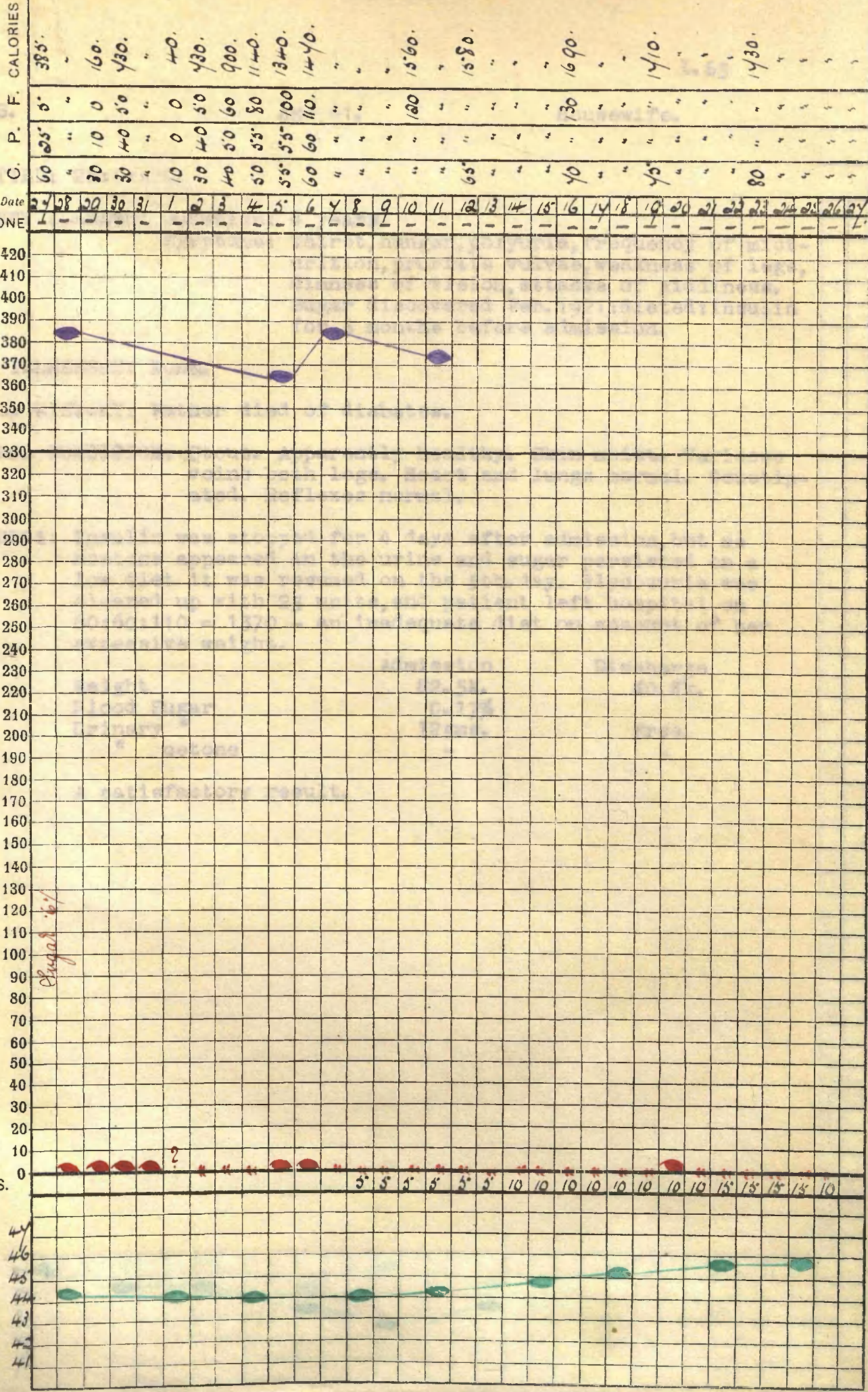
TOTAL URINARY SUGAR

Date of Admission...  
*27. X. 24*

Height  
*4 ft. 5 in.*

INSULIN UNITS.

WEIGHT IN KILOS





Mrs B.

Age 41.

Housewife.

ADMITTED: 26:11:24.

PRESENT ILLNESS: Duration: 4 years.

Symptoms: Thirst, hunger, polyuria, frequency of micturition, pruritis vulvae, weakness of legs, dimness of vision, attacks of giddiness.  
 Sugar discovered Feb. 1921: dieted: insulin for 3 months before admission.

PAST ILLNESSES: None.

FAMILY HISTORY: Father died of diabetes.

GENERAL CONDITION: Stout. Apparently healthy. Skin moist. Varicose veins both legs. Heart and lungs normal. Constipated. Reflexes normal.

PROGRESS: Insulin was stopped for 4 days after admission, but as acetone appeared in the urine and sugar persisted on a low diet it was resumed on the 5th. day. Glycosuria was cleared up with 25 units, and patient left hospital on 60:60:110 = 1370 - an inadequate diet on account of her excessive weight.

	Admission	Discharge
Weight	82.5k.	80.8k.
Blood Sugar	0.17%	
Urinary "	12gms.	Free.
" Acetone	-	-

A satisfactory result.



C. P. F. CALORIES

GRAMMES

## DIET

Mrs. Byrd.

Date	26	27	28	29	30	1	2	3	4	5	6	7	8	9	10	11	12	13	14
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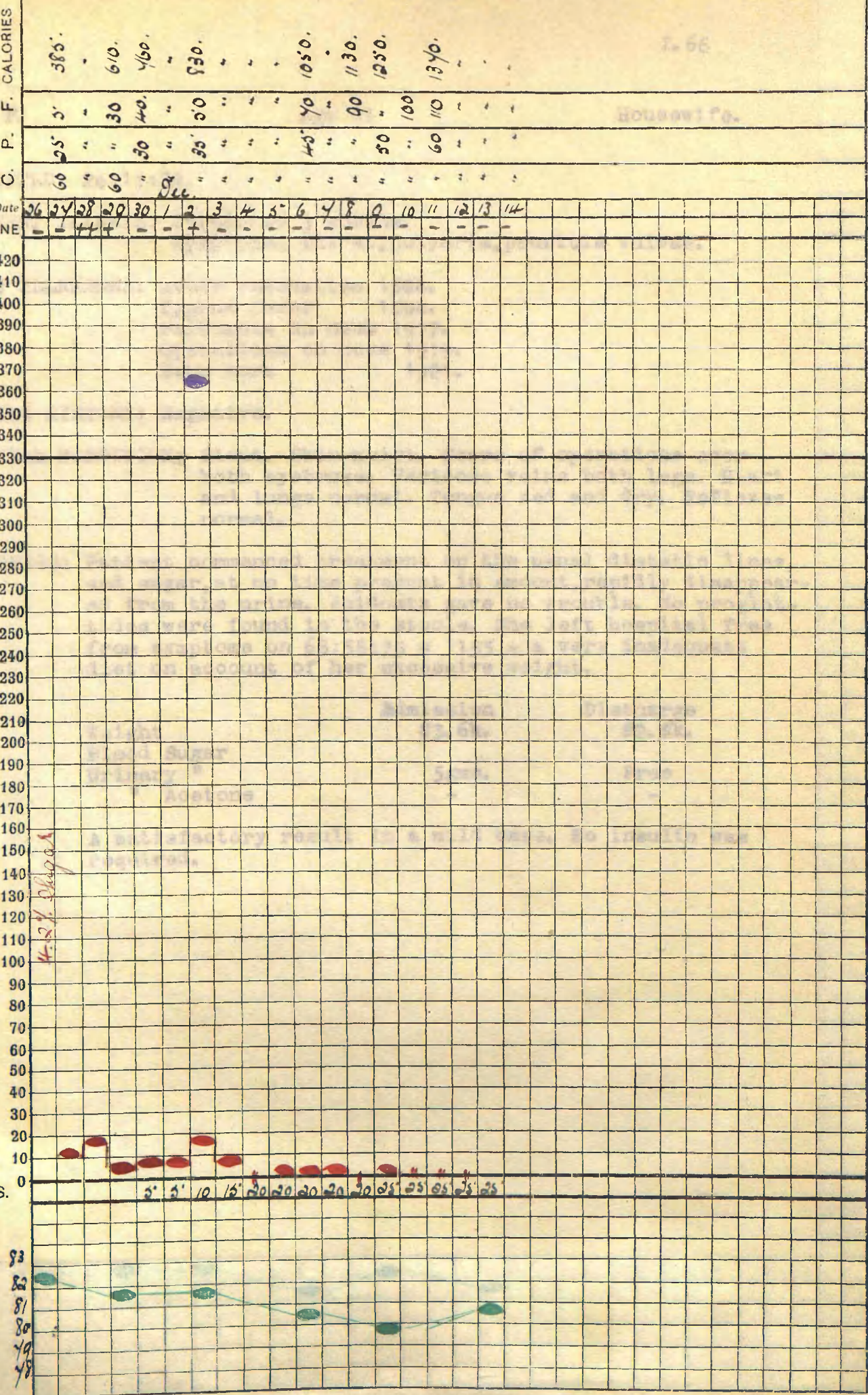
Name		ACETONE	
Age	41 yrs		
Ward	6	0-45	420
Journal	4	0-40	410
Page	6	0-35	400
		5-30	390
BLOOD	4	0-25	380
SUGAR	3	0-20	370
PER	2	0-15	360
CENT	1	0-10	350
		5-05	340

TOTAL  
URINARY  
SUGAR

Date of Admission...  
26/11/24

INSULIN UNITS.

WEIGHT  
IN KILOS





Mrs F.

Age 61

Housewife.

ADMITTED: 26:11:24.

PRESENT ILLNESS: Duration: 5 months.

Symptoms: Thirst, polyuria, pruritis vulvae.

PAST ILLNESSES: Acute rheumatism 1884.

Typhus fever 1894.

Carbuncle on neck 1917.

Operations on nose 1919.

Tape worm 1921.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Skin moist. Scars of operations over both eyebrows. Varicose veins both legs. Heart and lungs normal. Tongue red and dry. Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic lines, and sugar, at no time present in amount, rapidly disappeared from the urine. Acidosis gave no trouble. No proglottides were found in the stools. She left hospital free from symptoms on 65:55:75 = 1155 - a very inadequate diet on account of her excessive weight.

	Admission	Discharge
Weight	83.6k.	82.8k.
Blood Sugar		
Urinary "	5gms.	Free
" Acetone	-	-

A satisfactory result in a mild case. No insulin was required.







Mrs B.

Age 62.

Housewife.

ADMITTED: 26:11:24.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Thirst, polyuria, defective vision, numbness  
Rt. leg, ulcers Rt. foot, loss of power Rt. leg.  
Sugar discovered 1 year ago: dieted with  
little success.

PAST ILLNESSES: Phlegmasia alba dolens 1890.

Varicose ulcer Rt. leg 1912.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Heavily built. Muscles flabby. Skin dry and harsh.  
Gangrenous patch Rt. foot. Ulcer Rt. great toe.  
V.V. both legs. Rheumatoid condition joints Rt. leg.  
Heart normal: arteries not unduly thickened. Pupils  
react sluggishly to light. Knee jerks present.  
Tongue dry: constipated. Breath has odour of acetone.  
Urine contains a trace of albumen: sugar ++. Wass. neg.

PROGRESS: As acidosis was fairly marked and patient's general condition  
far from satisfactory insulin was administered on the 2nd.  
day. Patient improved somewhat, acetone and sugar disappear-  
ing from the urine with 40 units of insulin on an adequate  
diet. On 15:12:24, however, she developed a left hemiplegia.  
The temp. rose, the gangrene commenced to spread, and she was  
very ill. She was taken home by her relatives.

	Admission	Discharge
Weight	63k.	65.5k.
Blood Sugar	0.22%	0.23%
Urinary "	28gms.	Free.
" Acetone	++	-

An unsatisfactory result in a bed-ridden case of Rheumatoid  
Arthritis with mild diabetes. A terminal cerebral haemorrhage.

Subsequent Progress: Patient became gradually weaker, and died on  
13:1:25.



DIET  
GRAMMES  
G. P. F. CALORIES

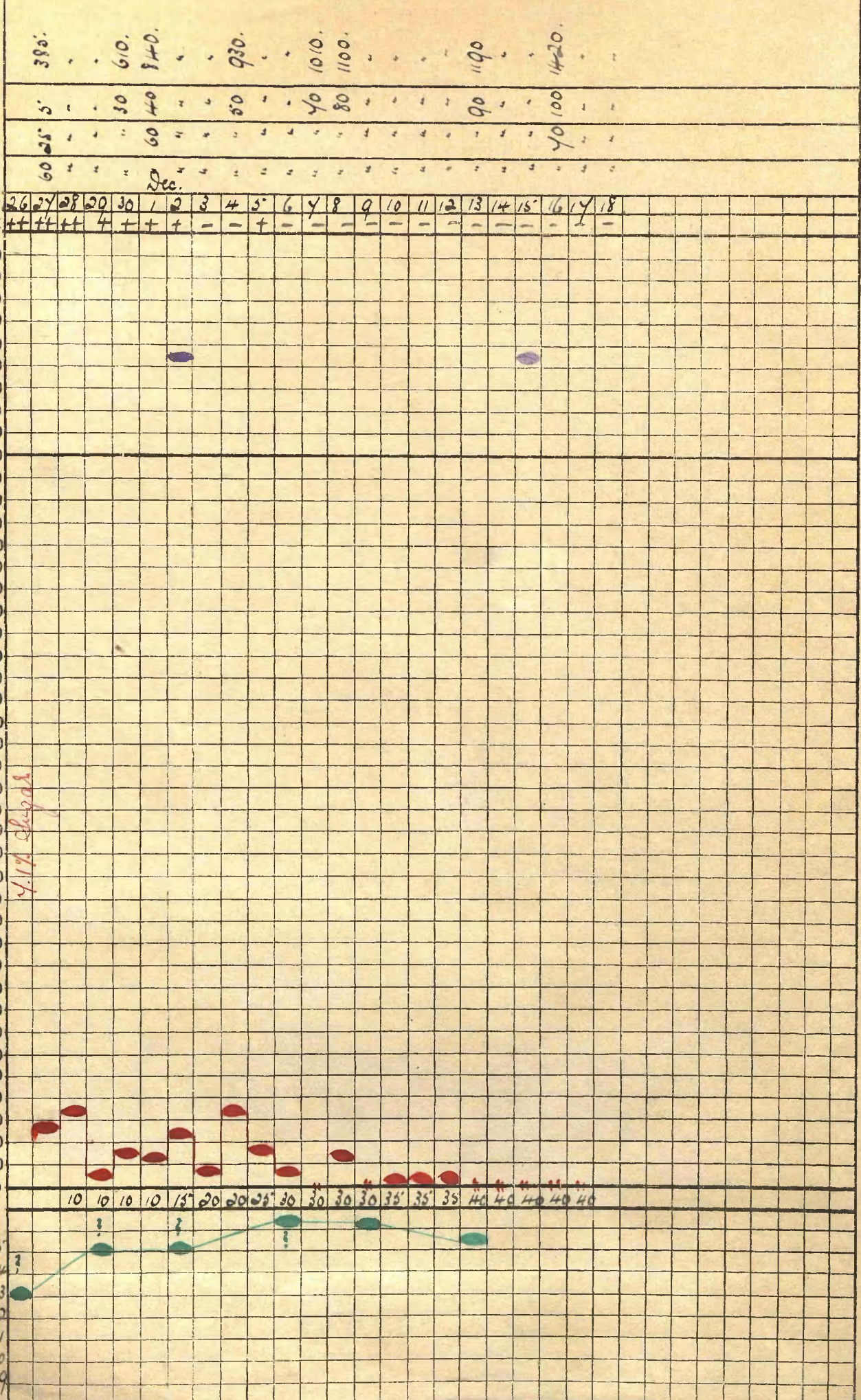
Name *Mrs. Bingham* Date *Dec.*  
Age *63* ACETONE  
Ward *6* 8 0.45 420  
Journal *6* 0.40 410  
Page *6* 0.35 400  
0.30 390  
BLOOD 0.25 380  
SUGAR 0.20 370  
PER 0.15 360  
CENT 0.10 350  
0.05 340

TOTAL  
URINARY  
SUGAR

Date of Admission...  
*26/11/24*

INSULIN UNITS.

WEIGHT  
IN KILOS



**SYNOPSIS OF CASES**

**'ALLEN' TREATMENT**

**MALE**

**A. 1 - P. 5.**

**FEMALE**

**A. 23 - P. 14.**



Case	Age	Dia.	Ren.	Lag.	Dia-.	Not D.	Ac.	Em.	Wt.	Remarks
A. 1	23	+					+		-5	
A. 2	10	+					+		0	Irreg. Dis.
A. 3	13	+					+		+2	
A. 4	36	+					+		0	Irreg. Dis.
A. 5	25	+					+		-4	
A. 6	32					+			0	
A. 7	38	+					+		-5	
A. 8	56	+							0	
A. 9	39	+							+4.5	
A. 10	27	+							-3	Phthysis.
A. 11	66					+			-3	Art. Scleros.
A. 12	57					+			0	Otitis Media.
A. 13	36	+					+		-2.5	
A. 14	58				+				0	Tabes Dorsalis.
A. 15	27	+							0	Irreg. Dis.
A. 16	49	+							-1.5	
A. 17	37	+					+		0	Pneumon. Death
A. 18	28	+							-4	
A. 19	43	+					+		-9	
A. 20	22	+							-1.5	
A. 21	17	+					+			Death.
A. 22	29	+					+			Irreg. Dis.
P. 1	42	+					+		-5	



Case	Age	Dia.	Ren.	Lag.	Dia-.	Not D.	Ac.	Em.	Wt.	Remarks.
P.2	13	+					+		-4	
P.3	38	+						+	0	
P.4	17	+							-2	
P.5	34	+							-6	
P.6	31	+							-1	
P.7	42	+					+		-3	
P.8	5	+							0	

[illegible]

J. C.

Age 23

Greengrocer.

ADMITTED: 24:10:19.

PRESENT ILLNESS: Duration: 5 weeks.

Symptoms: Thirst, polyuria, weakness, loss of weight

Sugar discovered 1 month ago.

PAST ILLNESSES: Operation for Glands in neck 1912.

Gassed, phosgene, 1918.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Tall: well built. Skin dry. Odour of acetone in breath: teeth septic. Heart and lungs normal. Reflexes normal.

PROGRESS: Patient was admitted before the Allen method of treatment had been introduced, and he was treated on the van Noorden lines with oatmeal and vegetables. Acidosis and glycosuria were very marked. No appreciable effect for 4 weeks, when there was a distinct fall in urinary acetone and sugar, and he was decidedly better. Improvement, however, was short-lived. On 9:12:19 he became very drowsy, vomited frequently, and the pulse became rapid and small. Intravenous saline was administered, 2 pints. This was followed by a rigor and great restlessness: but 24 hours later patient was much better and the drowsiness had completely passed off. Following this progress was not satisfactory, save that acidosis was less marked. On 10:1:20 Allen treatment was commenced and continued for 5 weeks. No serious attempt at starvation was made, and throughout the fat content of the diets was too high. None the less the acidosis became much less severe, and the sugar on the whole ran at a lower level. Patient left hospital on 70:85:175 = 2195.

	Admission	Discharge
Weight	58.2k.	53.2k.
Urinary Sugar	170gms.	108gms.
Urinary Acetone	++++	++

This was the first case to be treated on Allen lines. The regime was not applied sufficiently rigorously. A case of severe diabetes with van unsatisfactory result. Patient died at home on July 1920.



[illegible]



A. F.

Age 10

Schoolboy.

ADMITTED: 13:12:49.

PRESENT ILLNESS: Duration: 2 weeks.

Symptoms: Thirst, polyuria, enuresis,  
emaciation, pains in head and back.

PAST ILLNESSES: Measles 1910.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished. Skin dry. Tongue dry: odour  
of acetone in breath. Heart and lungs  
normal. Reflexes normal.PROGRESS: As patient showed evidence of considerable acidosis  
treatment commenced with oatmeal diet. On this  
diet the acetonuria became decidedly less marked,  
but patient objected very strongly to the treatment  
and he was taken home by his parents on the 6 th.  
day.

	Admission	Discharge
Urinary Sugar	75gms.	27gms.
Urinary Acetone	+++	++

A case of severe diabetes, discharged irregularly.





S. McK.

Age 13

Schoolboy.

ADMITTED: 18:12:19.

PRESENT ILLNESS: Duration: 7 days.  
Symptoms : Thirst, polyuria.

PAST ILLNESSES: Scarlet Fever 1911.  
Influenza and Bronchitis 1918.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well developed. Skin moist. Tongue furred.  
Heart and lungs normal. Reflexes sluggish.  
A few glands palpable in neck.

PROGRESS: Treatment commenced with the Oatmeal diet of van Noorden, and this was continued for 25 days. Toward the end of this period the urine was sugar-free. On 13:1:20 treatment with Allen diet was started, and with increasing feeding there was no return of glycosuria, nor of acidosis although very large quantities of fat were given. Ultimately glycosuria reappeared but in small amount. At this time unfortunately the boy began to break diet, and thereafter treatment was impossible. He was discharged from hospital after numerous warnings on 40:60:175 = 1975 .

	Admission	Discharge
Weight	30k.	32k.
Urinary Sugar	45gms.	22gms.
Urinary Acetone	+	++

One of the first cases treated on 'Allen' Lines.  
The boy's behaviour rendered successful treatment impossible.







T. O.

Age 36

Baker.

ADMITTED: 20:1:20.

PRESENT ILLNESS: Duration: 3 months.

Symptoms: Thirst, polyuria, loss of weight,  
pains in abdomen.

PREVIOUS ILLNESSES: Nil.

FAMILY HISTORY: One brother had Phthisis.

GENERAL CONDITION: A deaf mute. Thin. Skin dry and slightly  
bronzed. Tongue dry: teeth coated with  
tartar. Reflexes normal.PROGRESS: On admission the urine contained acetone ++ and  
sugar 98 gms. in the first 24 hrs. Patient was  
placed upon the usual starvation dietary, but he  
refused to submit to the regime and left hospital  
irregularly on 22:1:20.



E. McL.

Age 25

Grocer.

ADMITTED: 16:2:20.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Thirst, frequency of micturition,  
headaches.Sugar discovered Jan. 1920: dieted with  
poor result.

PAST ILLNESSES: Measles in childhood.

Displaced Cartilage 1917.

Influenza 1918.

Boil on neck 1919.

FAMILY HISTORY: One sister suffered from bone Tubercle.

GENERAL CONDITION: Well built. Skin dry. Tongue dry and furred.  
Heart and lungs normal. Reflexes normal.

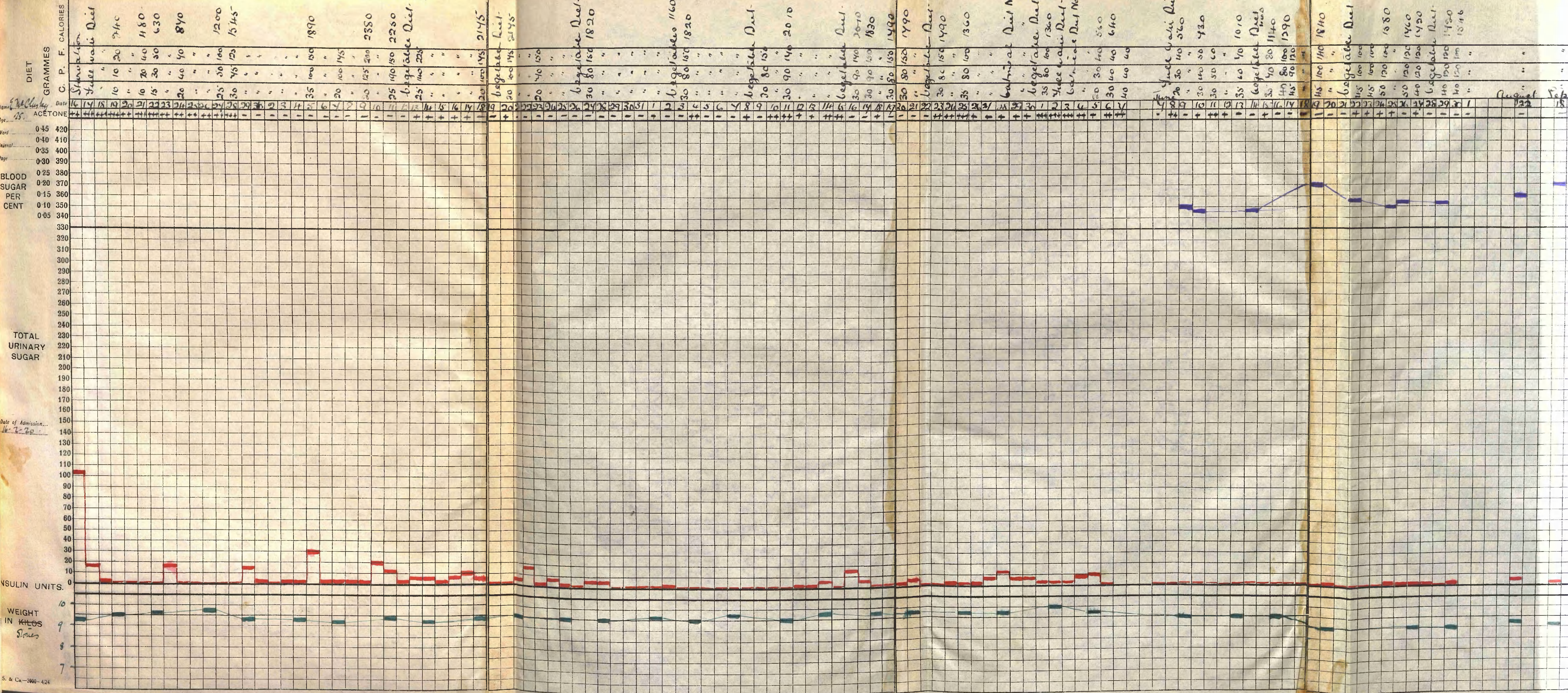
PROGRESS: Acidosis, marked on admission, was cleared up by starvation-dietary, while the urine became sugar-free. With increasing diet glycosuria returned and persisted, but subsequently it was found that patient was eating extra food. From time to time acetone appeared in the urine, but latterly, on strict diet, the urine was acetone and sugar free. The carbohydrate tolerance was low. Patient left hospital on 40:120:100  $\pm$  1540, free from symptoms.

Patient was seen frequently during the following year. He remained very well, maintaining his weight, though sugar was constantly present in the urine in small amount.

READMITTED: 13:1:21. Glycosuria had become very marked and there was acetone in the urine. Physical condition was unchanged. He responded very well to treatment, but but again it was found difficult to keep the urine sugar-free. Tolerance was little changed. He left hospital for domestic reasons on 35:90:110 = 1490 with slight glycosuria.

READMITTED: 10:1:22. Since dismissal in March 1921 he had dieted strictly and had been fairly well and able to work. But in Dec. 1921 he had to give up on account of weakness, and latterly he had felt very drowsy. On admission patient was sleepy and dull. The breath had a strong odour of acetone. There was considerable generalised oedema. The abdomen was distended and





DIET  
GRAMMES  
P. F. CALORIES  
C.

Word 0.45 420  
Journal 0.40 410  
Page 0.35 400  
0.30 390  
0.25 380  
BLOOD 0.20 370  
SUGAR 0.15 360  
PER 0.10 350  
CENT 0.05 340

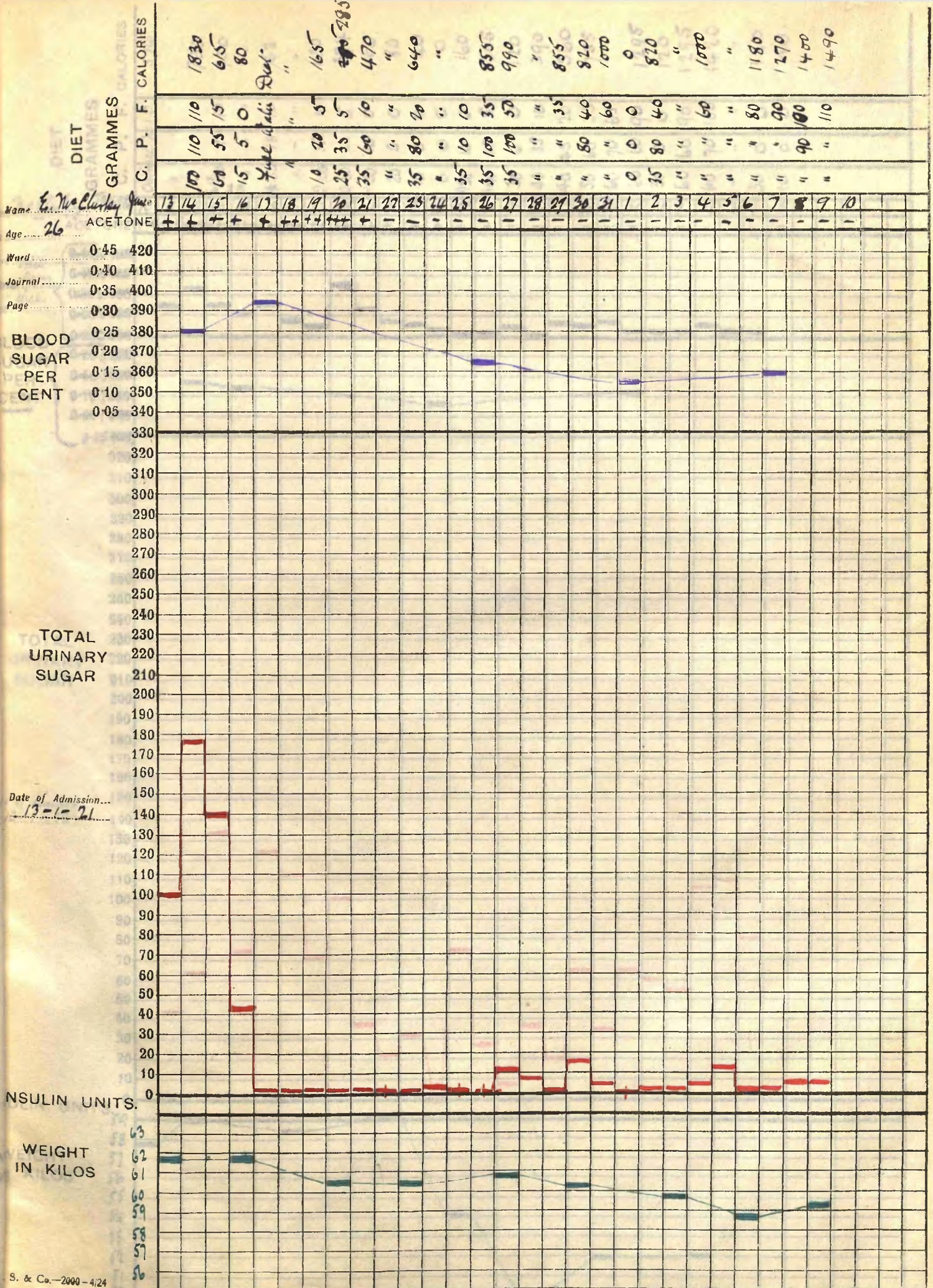
TOTAL  
URINARY  
SUGAR

Date of Admission...  
10-7-20

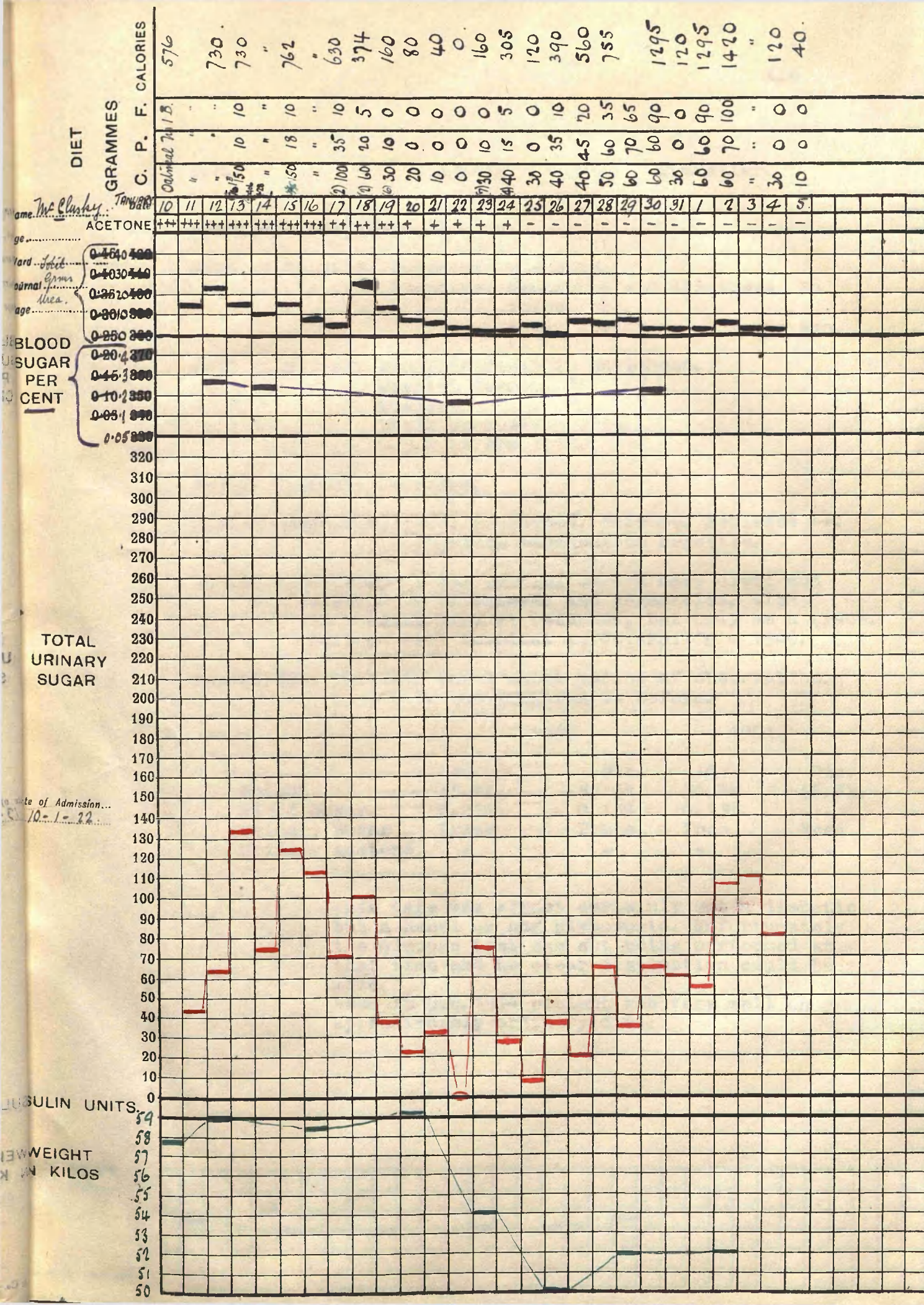
INSULIN UNITS.

WEIGHT  
IN KILOS  
Stones









T.R.

Age 32

Traveller.

ADMITTED: 10:7:20.

PRESENT ILLNESS: Duration: 9 months.

Symptoms: Headaches and dizziness. No  
thirst etc.

Sugar discovered 1 month ago.

PAST ILLNESSES: Whooping Cough in childhood.

Scarlet Fever " "

Measles " "

Shell Concussion 1916.

G. S. W. Rt. Arm 1917.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished. Nervous. Reflexes ++.  
Physical examination negative.PROGRESS: Glycosuria was trivial on ordinary diet, and  
cleared up on oatmeal and vegetables. With  
increasing diet it returned, but only as a trace.  
Patient left hospital on 40:120:100 = 1540.READMITTED: 29:1:21. For a short period of observation.  
Condition and progress as before.

1920

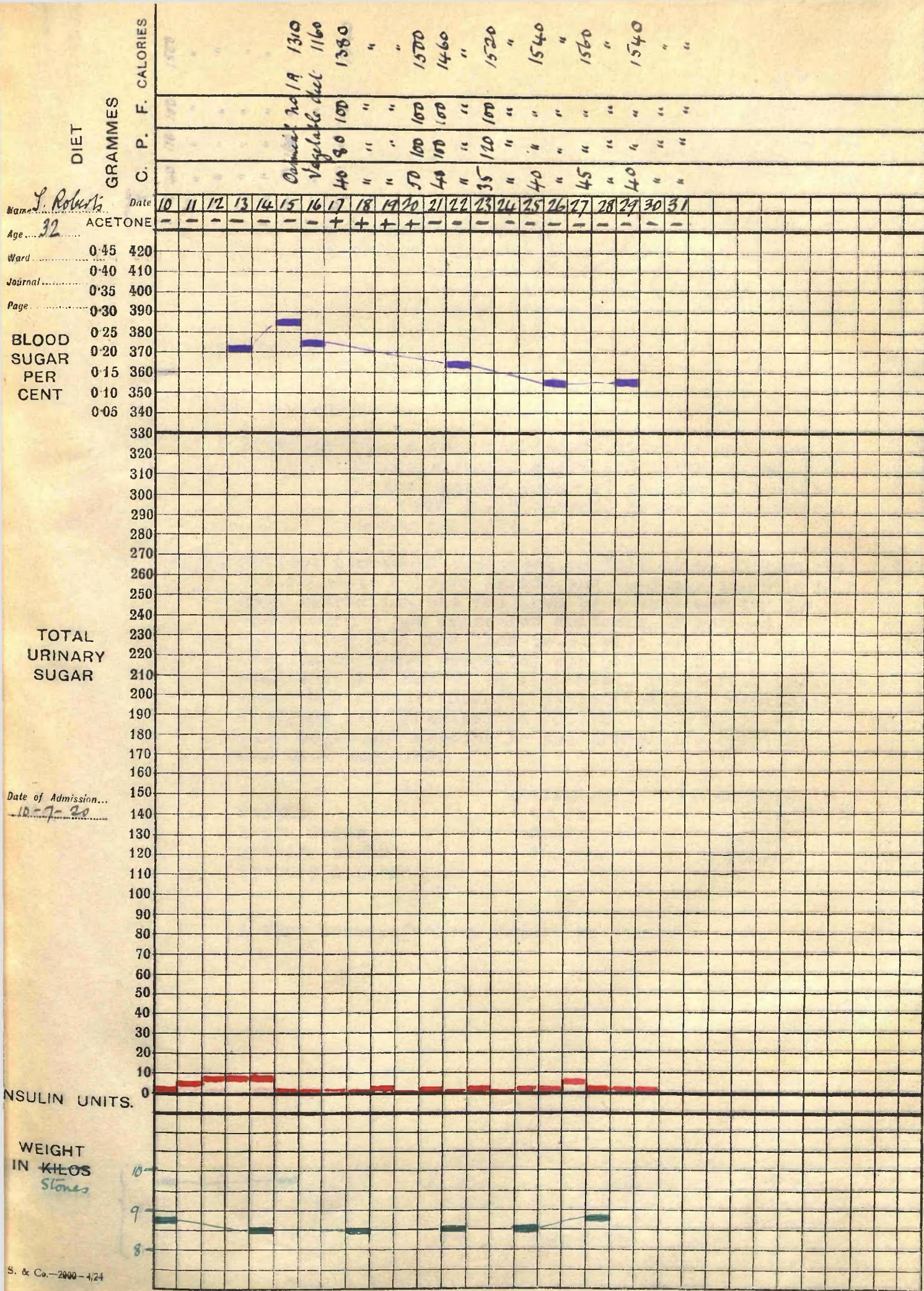
1921.

	Ad.	Dis.	Ad.	Dis.
Weight	55.4k.	55.5k.	56.8k.	56.8k.
Blood Sugar	0.21%	0.13%	0.13%	-
Urinary Sugar	Trace	Trace	Free	Free
Urinary Acetone	-	-	-	-

This case was almost certainly not a diabetic but a Renal or Lag glycosuria. Unfortunately the Glucose test was not being performed at that time and no clear distinction could be made.

Seen in Dec. 1924 patient was very well on approximately ordinary diet.







C.	P.	F.	CALORIES
40	110	100	1500
"	"	"	"
"	"	"	"
"	"	"	"
"	"	"	"
50	160	"	"
"	"	"	"
60	"	"	1570

Name T. Roberts Jan.

Age 32 ACETONE

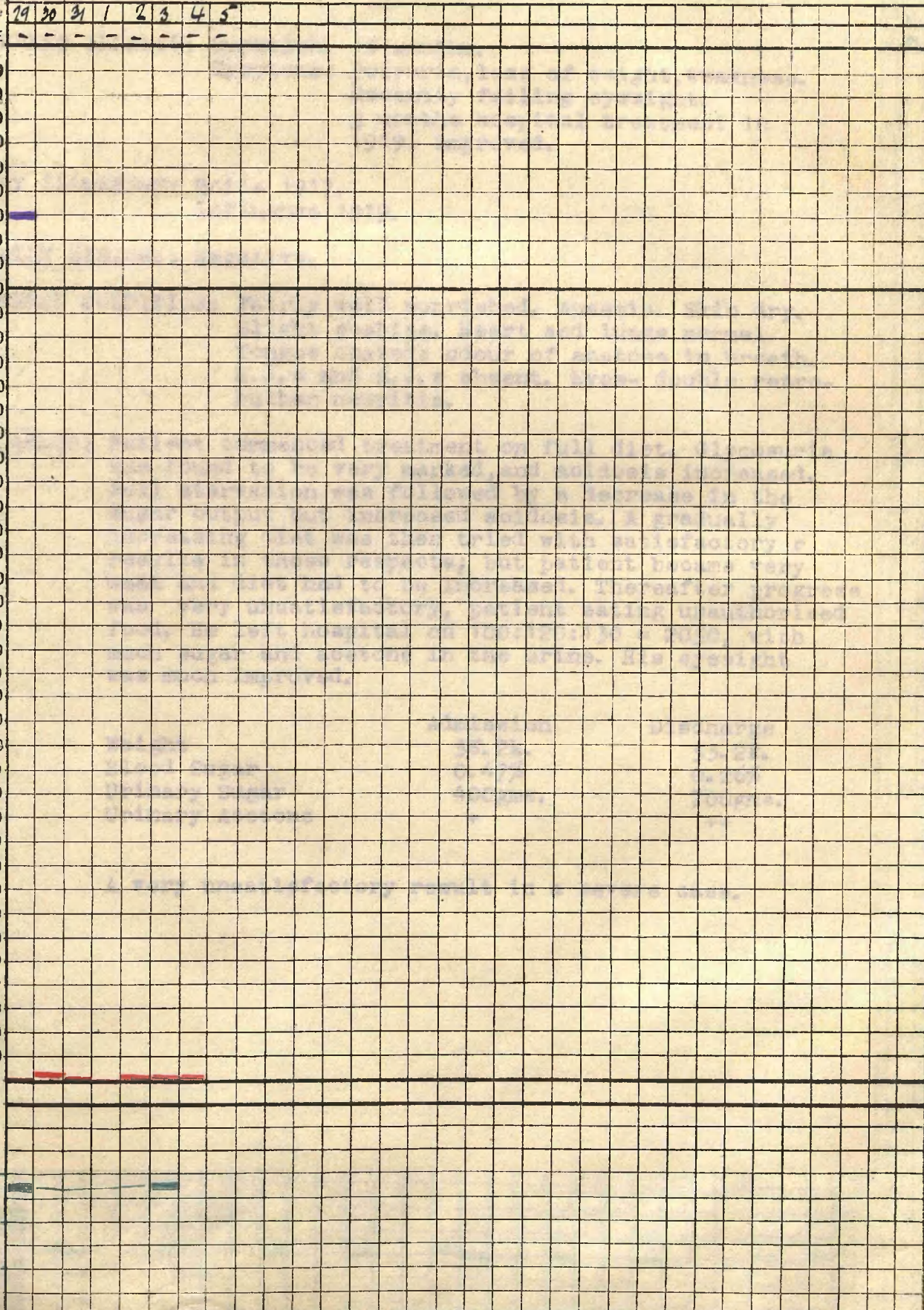
Ward .....	0-45	420
Journal .....	0-40	410
Page .....	0-35	400
	0-30	390
BLOOD	0-25	380
SUGAR	0-20	370
PER	0-15	360
CENT	0-10	350
	0-05	340

TOTAL  
URINARY  
SUGAR

Date of Admission.. 19-1-21

INSULIN UNITS.

WEIGHT  
IN ~~KILOS~~  
Stones



J.O'D.

Age 38

Steel Worker.

ADMITTED: 2:8:20.

PRESENT ILLNESS: Duration: 18 months.

Symptoms: Polyuria, loss of weight, weakness.  
 Recently failing eyesight.  
 5 months hospital treatment in  
 1919: improved.

PAST ILLNESSES: Boils 1917.  
 Influenza 1919.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Fairly well nourished. Anaemic. Skin dry.  
 Slight scabies. Heart and lungs normal.  
 Tongue coated: odour of acetone in breath.  
 K.J.s and A.J.s absent. Eyes- double retro-  
 bulbar neuritis.

PROGRESS: Patient commenced treatment on full diet. Glycosuria was found to be very marked, and acidosis increased. Full starvation was followed by a decrease in the sugar output but increased acidosis. A gradually decreasing diet was then tried with satisfactory results in these respects; but patient became very weak and diet had to be increased. Thereafter progress was very unsatisfactory, patient eating unauthorised food. He left hospital on 100:120:130 = 2050, with much sugar and acetone in the urine. His eyesight was much improved.

	Admission	Discharge
Weight	58.2k.	53.2k.
Blood Sugar	0.47%	0.26%
Urinary Sugar	400gms.	700gms.
Urinary Acetone	+	++

A very unsatisfactory result in a severe case.











E. G. *Goldman*

Age 56

Joiner.

ADMITTED: 12:11:20.

PRESENT ILLNESS: Duration: 6 years.

SYMPTOMS: Paraesthesia legs and hands, pain in feet,  
oedema of feet. No thirst etc.  
Sugar discovered 4 years ago.

PAST ILLNESSES: General oedema for some months 1913.

" " " " " 1916. No work since.

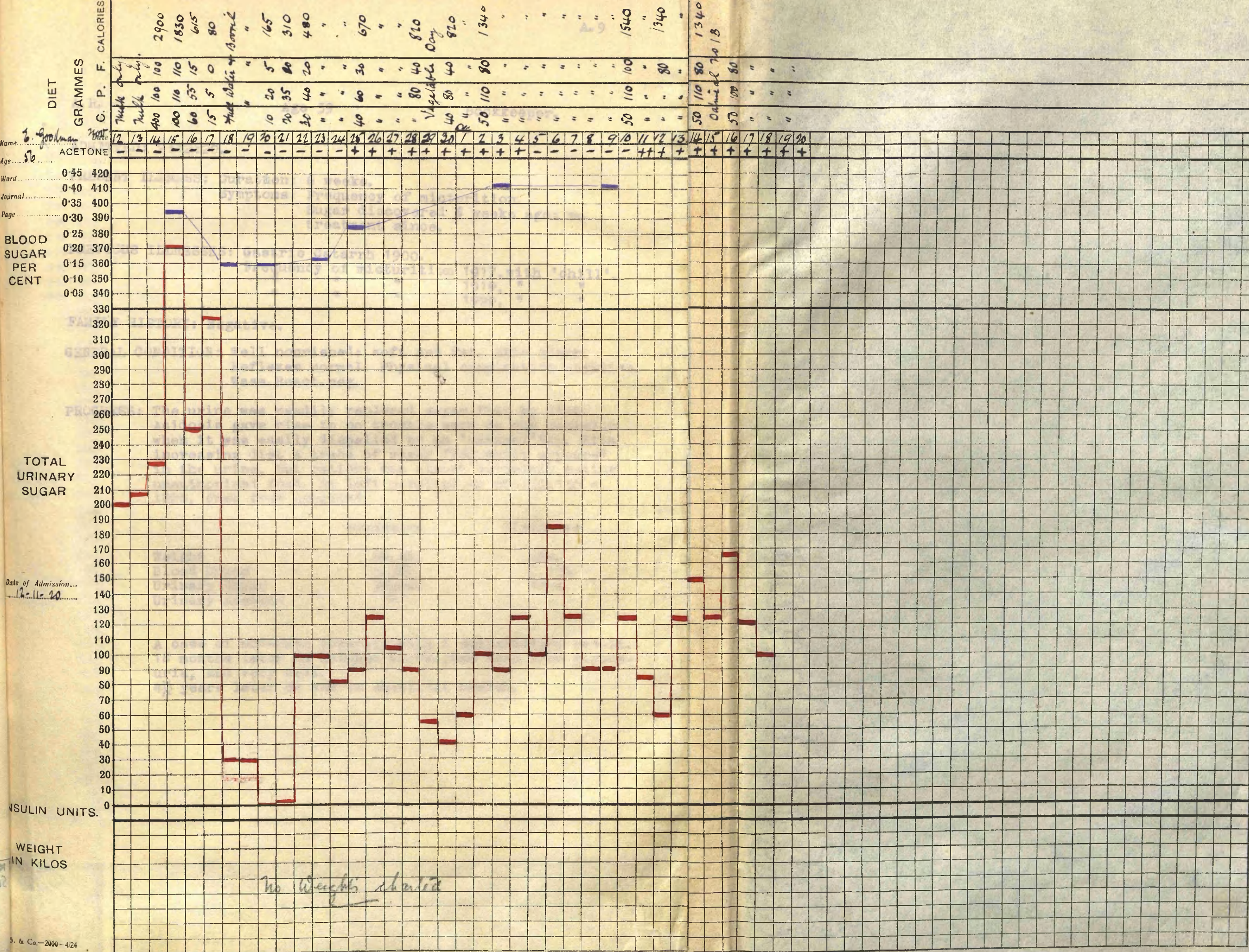
GENERAL CONDITION: Pale: anaemic: fairly well nourished. Generalised oedema. Gangrenous patch 4th. left toe: ulcer. Cellulitis left foot. Heart enlarged: V.S. mitral murmur. Arteries sclerosed: B.P. 160. Lungs- rale both bases. Tongue furred. Knee jerks and ankle jerks not elicited. Incipient cataract.

PROGRESS: Patient was very weak and strict dieting was found to be impossible. Nevertheless with 2 days' starvation the urine was rendered sugar-free. With resumption of feeding sugar returned in amount. Blindness increased, and was almost complete on dismissal. Gangrene to involve 4th. toe and a small area dorsum of foot. Oedema decreased, but did not disappear. Rale at bases increased in amount. He was taken home on 19:12:20, and died in Jan. 1921.

	Admission	Discharge
Blood Sugar	0.31%	0.41%
Urinary Sugar	200gms.	100gms.
Urinary Acetone	-	+

An unsatisfactory result in a severe case.







A. R.

Age 39

Bookkeeper.

ADMITTED: 2:12:20.

PRESENT ILLNESS: Duration: 6 weeks.

Symptoms: Frequency of micturition.

Sugar discovered 6 weeks ago: no treatment since.

PREVIOUS ILLNESSES: Gastric Catarrh 1900.

Frequency of micturition	1917,	with 'chill'.
"	"	"
"	"	"
"	"	"

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished: soft and fat. Skin clear.

Reflexes normal. Physical examination negative.

Wass. React. neg.

PROGRESS: The urine was readily rendered sugar-free by diet.

Acidosis gave rise to no trouble save on one occasion when it was easily dispelled by an 'oatmeal' day. With increasing diet a trace of sugar frequently appeared in the urine, but patient was almost certainly eating unauthorised food. He left hospital on 60:130:130 = 1820, free from symptoms.

	Admission	Discharge
Weight	64.5k.	69k.
Blood Sugar	0.08%	0.10%
Urinary Sugar	30gms.	Free.
Urinary Acetone	+	-

A case of moderate severity with a satisfactory result. 18 months later he weighed 61.4k., was free from glycosuria, and very well.

4½ years later he was in excellent health.







W. R.

Age 27

Clerk.

ADMITTED: 21:1:21.

PRESENT ILLNESS: Duration: 3 months.

Symptoms: Thirst, polyuria, loss of weight, weakness.

Sugar discovered 1 month ago.

PAST ILLNESSES: Whooping cough in childhood.

Right pleurisy 1912.

G. S. W. Left Chest, penetrating, 1917.

FAMILY HISTORY: Mother died of Phthisis.

GENERAL CONDITION: Poorly nourished. Scars of through and through wound left chest. Skin moist. Heart normal.

Lungs-evidence of consolidation right apex: some moist rale. Reflexes normal. Wass. React. negative.

PROGRESS: Sugar was quickly cleared from the urine by diet, but reappeared on low diet. An occasional starve-day or 'half-day' kept the glycosuria in check. Unfortunately the lesion at the Rt. apex began to spread (much rale and friction) and the Lt. apex became affected. Weight was lost and cough was very troublesome. Sugar tolerance was very low, but it was deemed advisable to increase diet and permit a slight glycosuria. He went home on 45:110:13 = 1790, far from well.

	Admission	Discharge
Weight	52k.	49.8k.
Blood Sugar	0.14%	0.18%
Urinary Sugar	105gms.	38gms.
Urinary Acetone	+	+

An unsatisfactory result in a fairly severe case of diabetes with phthisis.







W.C.

Age 66

Miner.

ADMITTED: 25:2:21.

PRESENT ILLNESS: Duration: 3 months.

Symptoms: Pain in metatarso-phalangeal joint  
left great toe. No thirst, polyuria,  
or loss of weight.

PAST ILLNESSES: Jaundice 1897.

Amputation Rt. lower leg for gangrene Rt. foot.

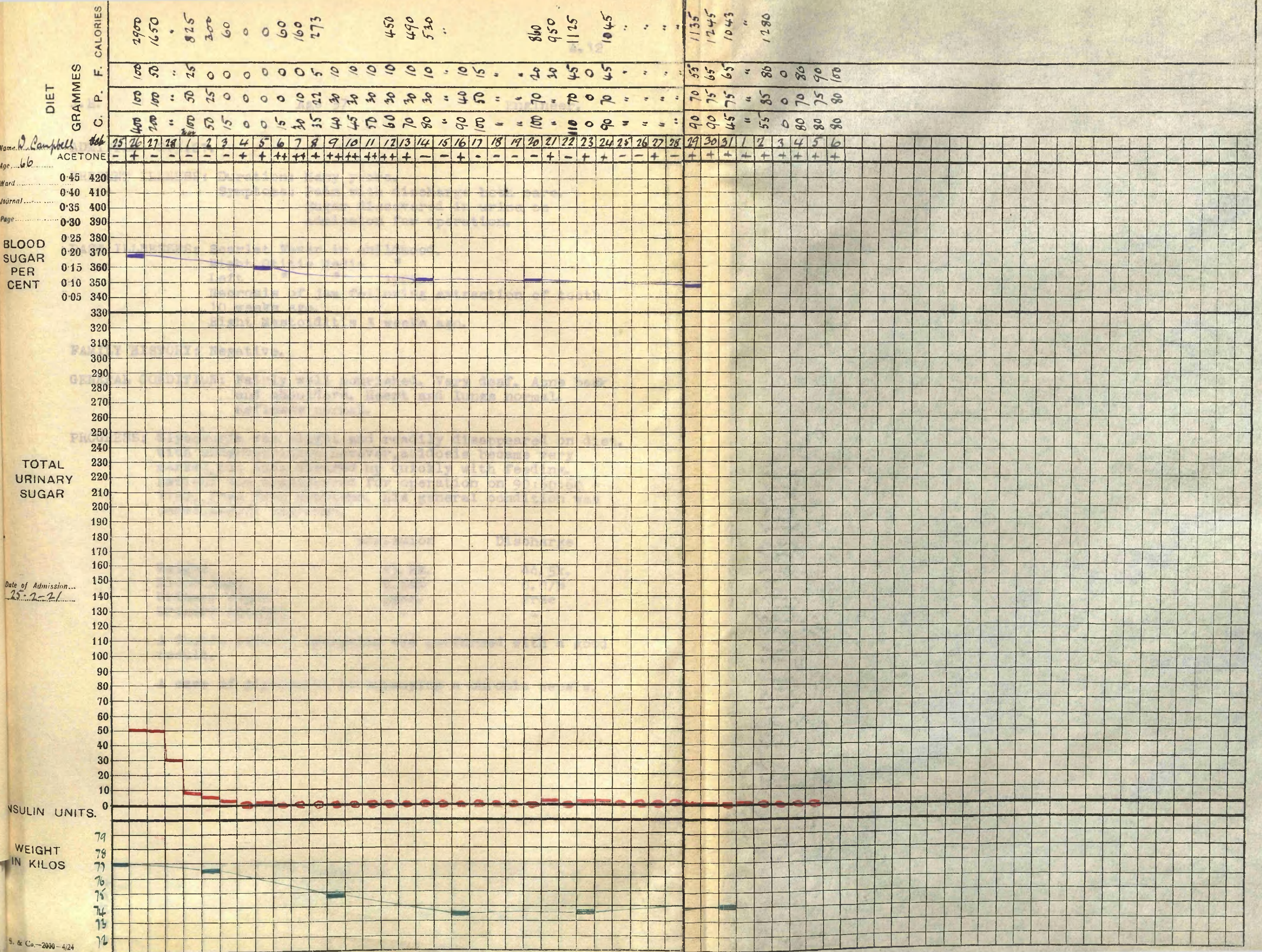
FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout: well nourished: florid. Rt. leg amput-  
ated. Left foot cyanosed: skin glazed: "scabs"  
left gt. toe. Heart enlarged: B.P. 155 mm. Hg.  
Lungs- rale both bases. Teeth septic.PROGRESS: Progress was uninterrupted. The glycosuria was rapidly  
cleared up by diet and recurred subsequently only in  
very small amount. Acetonuria followed starvation, but  
disappeared with increasing feeding. The left foot  
improved very much, and patient was able to get up  
and about when he went home. The diet on discharge was  
80:80:100 ± 1540.

A	Admission	Discharge
Weight	77k.	74k.
Blood Sugar	50gms.	Free
Urinary Sugar	0.18%	0.09%
Urinary Acetone	-	-

A case of glycosuria accompanying senile gangrene.







J. H.

Age 57

Engineer.

ADMITTED: 8:3:21.

PRESENT ILLNESS: Duration: Many years.

Symptoms: Pain with discharge both ears.

Sugar discovered in urine on admission for operation.

PAST ILLNESSES: Scarlet Fever in childhood.

Right Otitis Media "

Left " " 1908.

Necrosis of jaw following extraction of tooth 10 weeks ago.

Right Mastoiditis 3 weeks ago.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Fairly well nourished. Very deaf. Acne back and shoulders. Heart and lungs normal. Reflexes normal.

PROGRESS: Glycosuria was slight and readily disappeared on diet. With undernutrition, however, acidosis became very marked, but this cleared up quickly with feeding. Patient was transferred for operation on 90:60:60 = 1190, free from symptoms. His general condition was considerably improved.

	Admission	Discharge
Weight	45.2k.	44.5k.
Blood Sugar	0.08%	0.07%
Urinary Sugar	5gms.	Free
Urinary Acetone	-	-

A double mastoid operation was performed with a good result.

A case of glycosuria accompanying a chronic sepsis.







W. J. S. T.

Age 36

Steel-worker.

ADMITTED: 13:10:21.

PRESENT ILLNESS: Duration: 2½ years.

Symptoms: Polyuria, loss of weight, pains in back and legs.

Sugar discovered 1921: dieted with care: unable to dispel symptoms.

PAST ILLNESSES: Tonsillitis 1916.

Rheumatic Fever 1916.

Perineal Abscess 1916.

FAMILY HISTORY: One brother died of Phthisis. Otherwise negative.

GENERAL CONDITION: Tall: thin: poor condition. Skin dry: acne shoulders and back. Tongue furred, inflamed, and smooth. Physical examination otherwise negative.

PROGRESS: Patient commenced treatment on the usual dietetic lines and sugar was rapidly cleared from the urine. Acidosis lessened in amount, but did not entirely disappear even with increasing diet. Before an equilibrium diet could be reached patient had to return home for domestic reasons.

	Admission	Discharge
Weight	53.7k.	51.2k.
Blood Sugar	0.60%	0.27%
Urinary Sugar	158gms.	8gms.
Urinary Acetone	++	+

An unsatisfactory termination in a fairly severe case.



Name W. Thomas 824

Age, 36

Ward	0-43	420
	0-40	410

Journal .....	0.25	400
---------------	------	-----

Page ..... 1.24 1390

BLOOD ~~0.25~~ 1380  
~~0.20~~ 1370

SUGAR 0.45 360  
PER 0.45 360

PER  
CENT 0-10-2350

0-06-1340

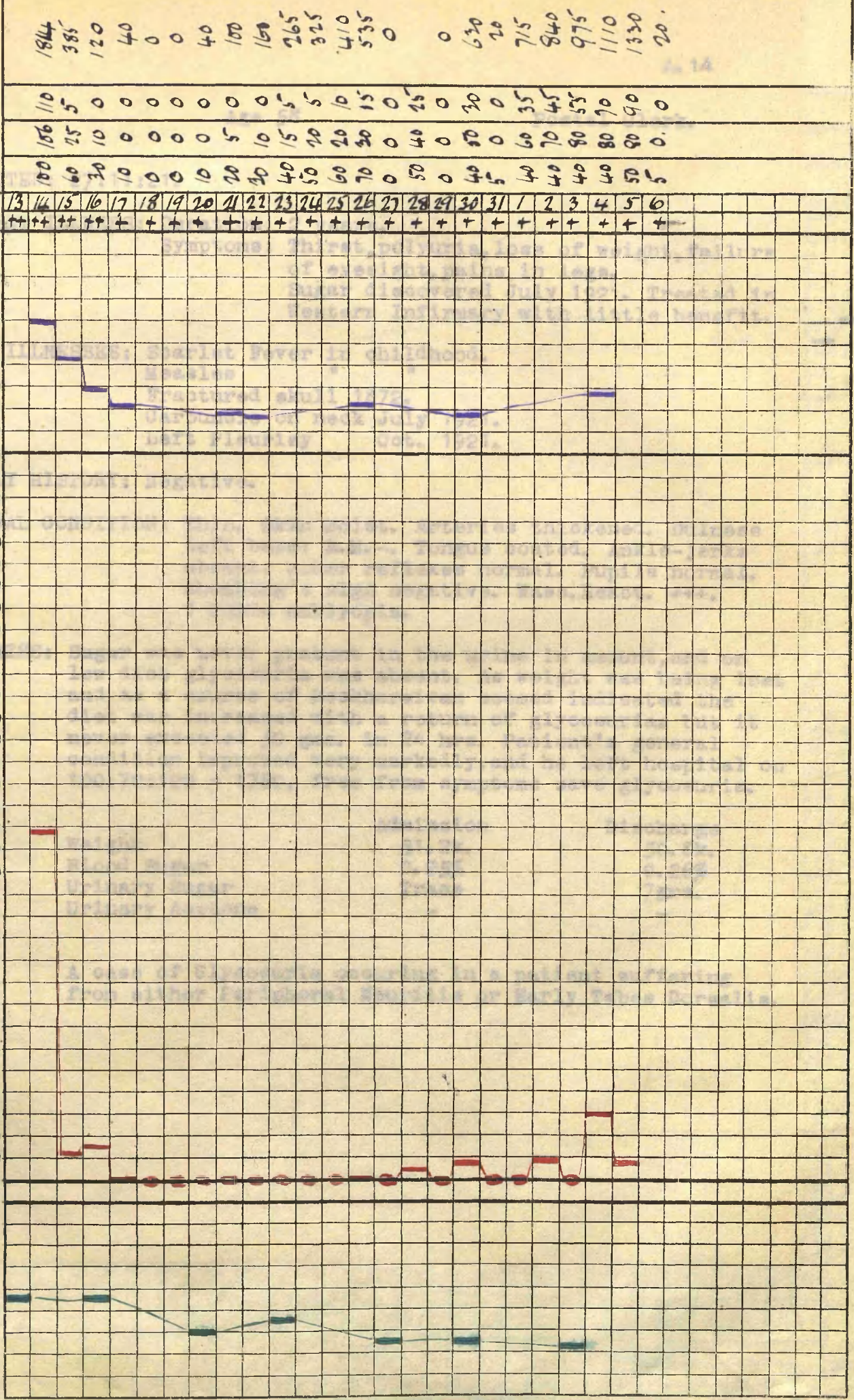
330  
320

TOTAL	230
URINARY	220
SUGAR	210

Date of Admission... 13-10-21

INSULIN UNITS. <sup>0</sup>

WEIGHT  
IN KILOS





W.P.

Age 58

Postal Clerk.

ADMITTED: 27:11:21.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, loss of weight, failure of eyesight, pains in legs.

Sugar discovered July 1921. Treated in Western Infirmary with little benefit.

PAST ILLNESSES: Scarlet Fever in childhood.

Measles " "

Fractured skull 1872.

Carbuncle on neck July 1921.

Left Pleurisy Oct. 1921.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin. Skin moist. Arteries thickened. Dulness left base: R.M.-. Tongue coated. Ankle-jerks absent: other reflexes normal. Pupils normal. Rhomberg's sign negative. Wass.React. +++.

? Toxic amblyopia.

PROGRESS: Sugar was never present in the urine in amount, and on low diet glycosuria was absent. As weight was being lost and as a course of Neokharsivan seemed indicated the diet was increased with a return of glycosuria: but it never exceeded 30 gms. in 24 hrs. Patient's general condition improved very markedly, and he left hospital on 100:70:120 = 1760, free from symptoms save glycosuria.

	Admission	Discharge
Weight	51.2k.	50.8k.
Blood Sugar	0.25%	0.26%
Urinary Sugar	Trace	7gms.
Urinary Acetone	-	-

A case of Glycosuria occurring in a patient suffering from either Peripheral Neuritis or Early Tabes Dorsalis.







J.B.

Age 27

Clerk.

ADMITTED: 22:12:21.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, incontinence of urine, emaciation, weakness.

PAST ILLNESSES: Compound Fracture Rt. Arm 1912.

Syphilis ( 606 treatment ) 1914.

Exophthalmic Goitre 1918.

Excision of Thyroid 1919.

FAMILY HISTORY: Good.

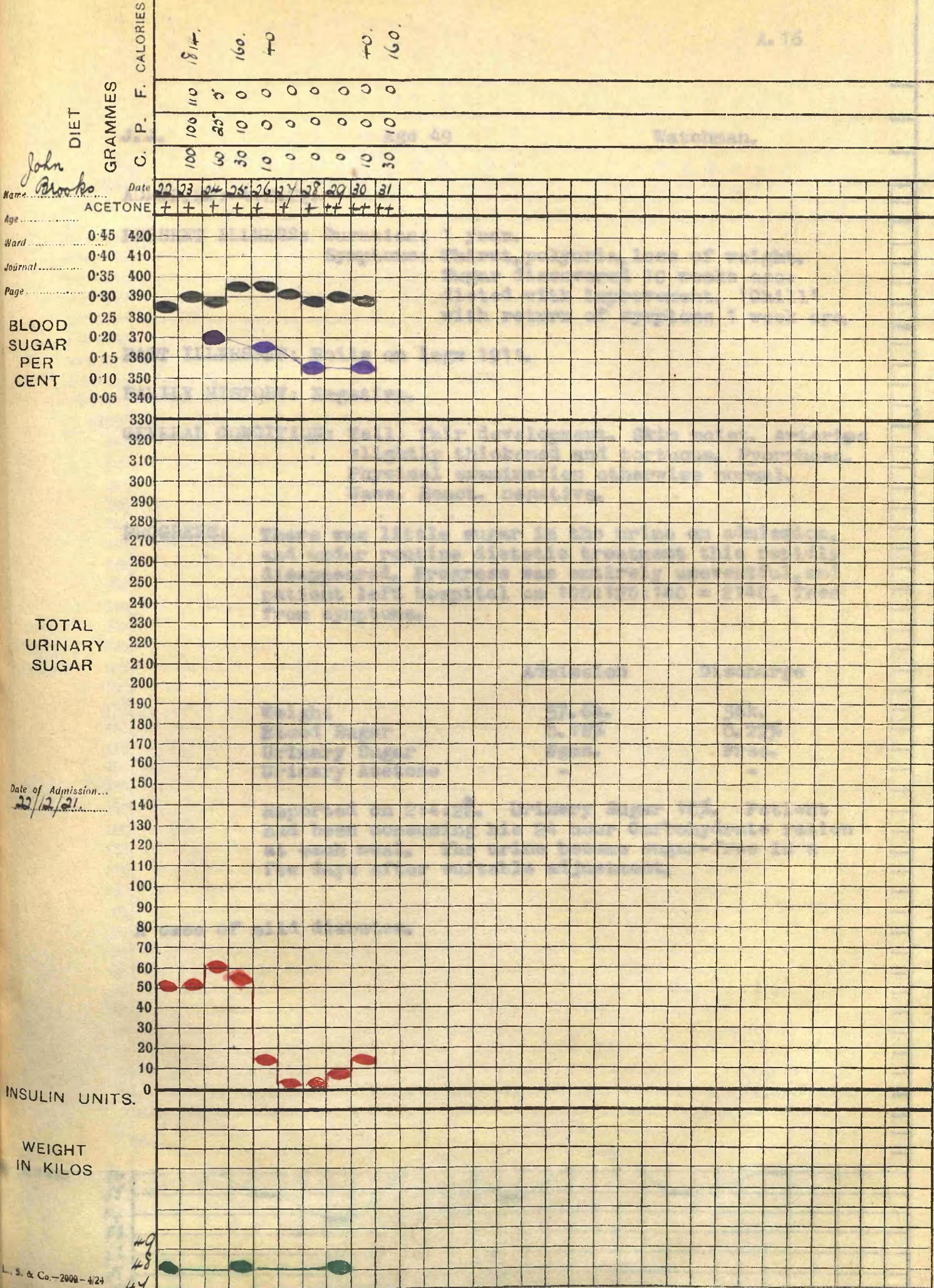
GENERAL CONDITION: Small. Well developed. Skin dry. ? some myxoedema. Rt. elbow ankylosed. Heart and lungs normal. Reflexes normal. Some bronchitis. Wass.React. negative. Teeth septic.

PROGRESS: Patient commenced treatment on the usual dietetic lines and the urinary sugar was rapidly reduced to a trace. But patient became dissatisfied with his treatment on the 10th. day and left hospital irregularly. The diet on the last day was 30:10:0 = 160.

	Admission	Discharge
Weight	48k.	48k.
Blood Sugar	0.20%	0.12%
Urinary Sugar	50 gms.	15 gms.
Urinary Acetone	+	++

A case of moderately severe diabetes. Treatment refused.







J. J.

Age 49

Watchman.

ADMITTED: 4:2:22.

PRESENT ILLNESS: Duration: 1 year.

Symptoms: Thirst, polyuria, loss of weight.  
 Sugar discovered 10 weeks ago:  
 dieted with improvement. 'Chill'  
 with return of symptoms 1 week ago.

PAST ILLNESSES: Boils on legs 1911.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Tall: fair development. Skin moist. Arteries  
 slightly thickened and tortuous. Pyorrhoea.  
 Physical examination otherwise normal.  
 Wass. React. negative.

PROGRESS: There was little sugar in the urine on admission,  
 and under routine dietetic treatment this rapidly  
 disappeared. Progress was entirely uneventful, and  
 patient left hospital on 100:120:140 = 2140, free  
 from symptoms.

	Admission	Discharge
Weight	57.6k.	56k.
Blood Sugar	0.18%	0.22%
Urinary Sugar	8gms.	Free.
Urinary Acetone	-	-

Reported on 2:4:22. Urinary Sugar 10%. Patient  
 had been consuming his 24 hour Carbohydrate ration  
 at each meal. The urine became sugar-free in a  
 few days after suitable adjustment.

A case of mild diabetes.



Name: T. Johnson Date: 4-1-22

Age: 40 Sex: M Height: 5'6" Weight: 140 lbs  
Blood Sugar: 100 mg % Urea: 10 mg %

BLOOD SUGAR PER CENT  
0-45 100  
0-40 110  
0-35 120  
0-30 130  
0-25 140  
0-20 150  
0-15 160  
0-10 170  
0-05 180  
0-00 190

TOTAL URINARY SUGAR

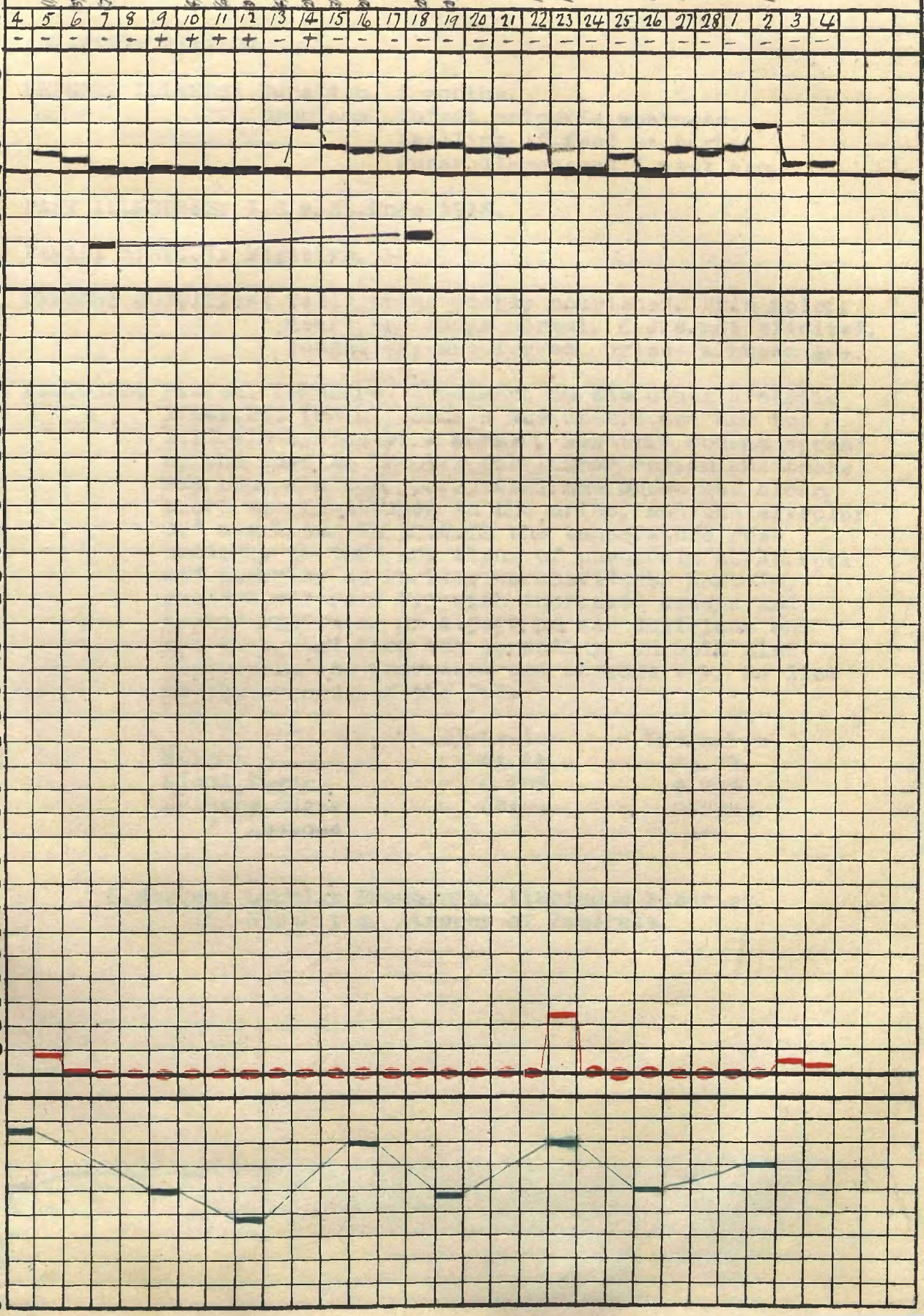
Date of Admission: 4-1-22

INSULIN UNITS

WEIGHT IN KILOS  
58  
57  
56  
55  
54  
53  
52  
51  
50

DIET  
GRAMMES  
C. P. F. CALORIES

1814	110	106	110	1814
385	5	25	5	385
140	0	10	0	140
40	0	0	0	40
40	0	0	0	40
100	0	5	0	100
140	0	10	0	140
305	5	15	5	305
326	5	20	5	326
410	10	20	10	410
535	15	30	15	535
705	25	40	25	705
0	0	0	0	0
830	30	50	30	830
955	35	60	35	955
1205	55	80	55	1205
1430	70	90	70	1430
1650	90	90	90	1650
1740	100	90	100	1740
1530	0	0	0	1530
1710	90	90	90	1710
1865	110	90	110	1865
2020	125	95	125	2020
2149	140	100	140	2149
	110	110		
	170	170		
	140			





M. S.

Age 37

Labourer.

ADMITTED: 21:4:22.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Thirst, polyuria, weakness,  
swelling of feet at night.  
Sugar discovered 1 week ago.

PAST ILLNESSES: G. S. W. Rt. Knee 1918.

FAMILY HISTORY: Negative.

PRESENT CONDITION: Tall: thin: poorly nourished. Skin moist.  
Heart and lungs normal. K.J.s not elicited.  
Tongue dry and furred. Urine- albumen ++.

PROGRESS: Patient commenced treatment on the usual dietetic lines, but the glycosuria was unaffected and the acidosis became more marked. Meantime oedema spread up the legs to involve the lumbar region, and there was some ascites. On 29:4:22 the chest was clear, there was no albumen in the urine, and the alveolar CO<sup>2</sup> was 4.3%. On 30:4:22 the temperature rose suddenly to 100° and signs of pneumonia at Rt. apex and pleurisy at Rt. base were evident. Next day patient was very ill with increased oedema and marked cyanosis. On 2:5:22 he was delirious and the lung condition was spreading. On milk diet glycosuria was increased and acetone +++. He died on the evening of the 2nd.

	Admission	Discharge
Weight	45.4k.	45.7k.
Blood Sugar	0.20%	0.25%
Urinary Sugar	68gms.	280gms.
" Acetone	+	+++

P. Mortem: Lobular Pneumonia, Fibrinous Pleurisy,  
Nephritis, Atrophy of Pancreas.



Name Mr. Smith Apals

Age 40

Weight 46

Normal Wt

Page 1

BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

Date of Admission 4-4-12

INSULIN UNITS.

WEIGHT IN KILOS

48  
47  
46  
45  
44  
43  
42  
41  
40

DIET

GRAMMES

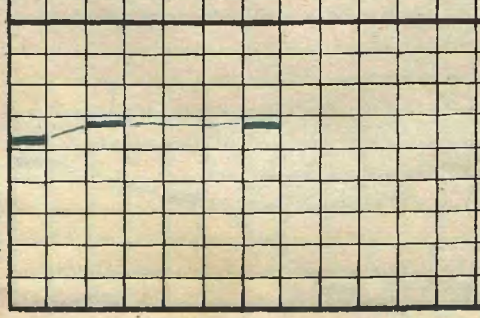
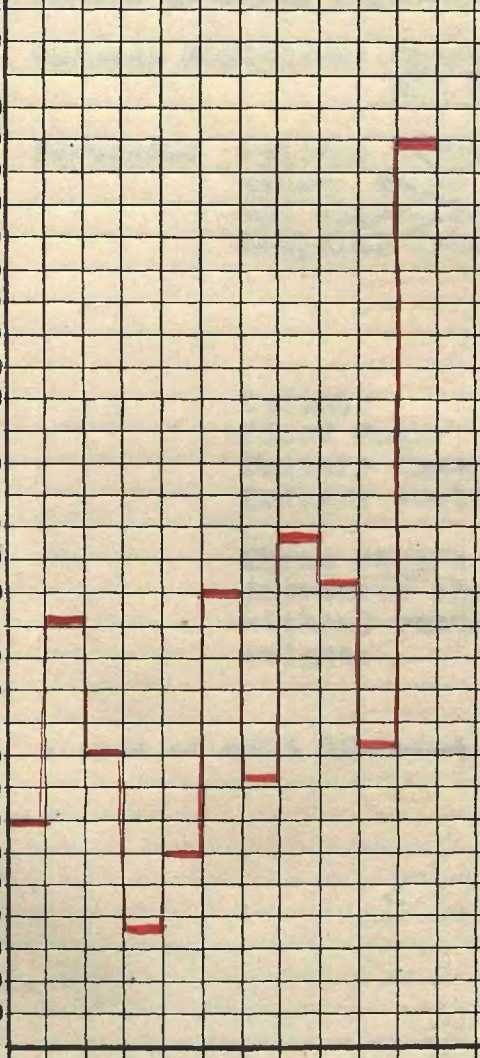
C. P. F. CALORIES

100 106 110 1814  
60 25 5 385  
10 0 160  
Oatmeal 700 13. 696  
" " " "  
" " " "  
150 10 10 730.  
" " " "  
100 10 0 440.  
Milk 2/66 cc + Sugar 70 gr  
1/2 Oatmeal 700 13 + Horlicks 1200 gr

21 22 23 24 25 26 27 28 29 30 1 2

ACETONE + + + + + + + + + + + + + + +

0.46 420  
0.40 410  
0.36 400  
0.30 390  
0.26 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340  
0.05 330





J.O.

Age 28

Warehouseman.

ADMITTED: 21:5:22.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Gradual loss of weight. Thirst  
and polyuria for 1 month.  
Sugar discovered 2 weeks ago.

PAST ILLNESSES: Measles in childhood.

Sunstroke 1915.

Gassed 1917.

Gastritis, ulcer, 1918: discharged from Army.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Spare build: emaciated. Skin moist. Heart  
and lungs normal. Reflexes normal.

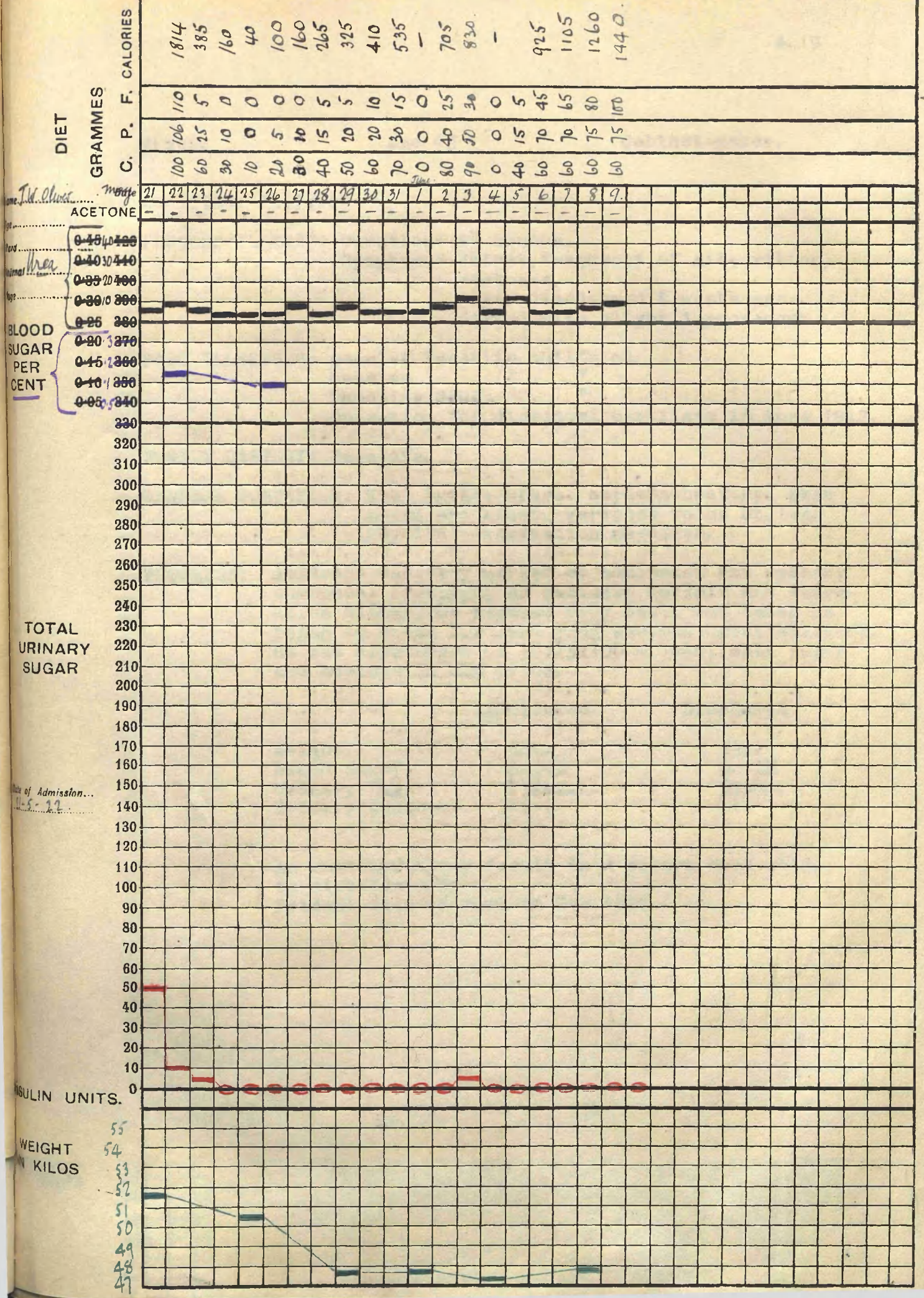
PROGRESS: Patient commenced treatment on the usual dietetic  
lines. Sugar rapidly disappeared from the urine,  
and thereafter progress was uninterrupted. He left  
hospital free from symptoms on 60:75:100 = 1440.

	Admission	Discharge
Weight	51.7k.	47.8k.
Blood Sugar	0.16%	0.10%
Urinary Sugar	51gms.	Free.
Urinary Acetone	-	-

Three months later ( Sept. 1922 ) patient had  
increased the Carbohydrate of his diet to 100gms.  
without return of glycosuria. He was gaining  
weight.

A case of mild diabetes.







W. McG.

Age 43

Cabinet-maker.

ADMITTED: 15:9:22.

PRESENT ILLNESS: Duration: 2½ months.

Symptoms: Thirst, frequency of micturition, weakness.

Sugar discovered 8 weeks ago:  
dieted with slight improvement.

PAST ILLNESSES: Scarlet Fever in childhood.

Measles " "

Whooping Cough " "

Operation for displaced cartilage in knee 1912.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well built: spare: appears healthy. Skin moist and clear. Varicose veins Rt. leg.  
Physical examination negative.

PROGRESS: Acidosis was very marked on admission and patient commenced treatment on oatmeal. To this and starvation dietary he reacted very well, but later he began to steal food, and progress was unsatisfactory. He was discharged on 50:85:100 = 1440, with sugar and acetone in the urine.

	Admission	Discharge
Weight	68k.	59k.
Blood Sugar	0.34%	0.12%
Urinary Sugar	75gms.	55gms.
Urinary Acetone	++++	+

An unsatisfactory result in a severe case owing to misbehaviour.  
Patient died in coma in Dec. 1922.



Date of Admission...	150
15/9/22	140
	130

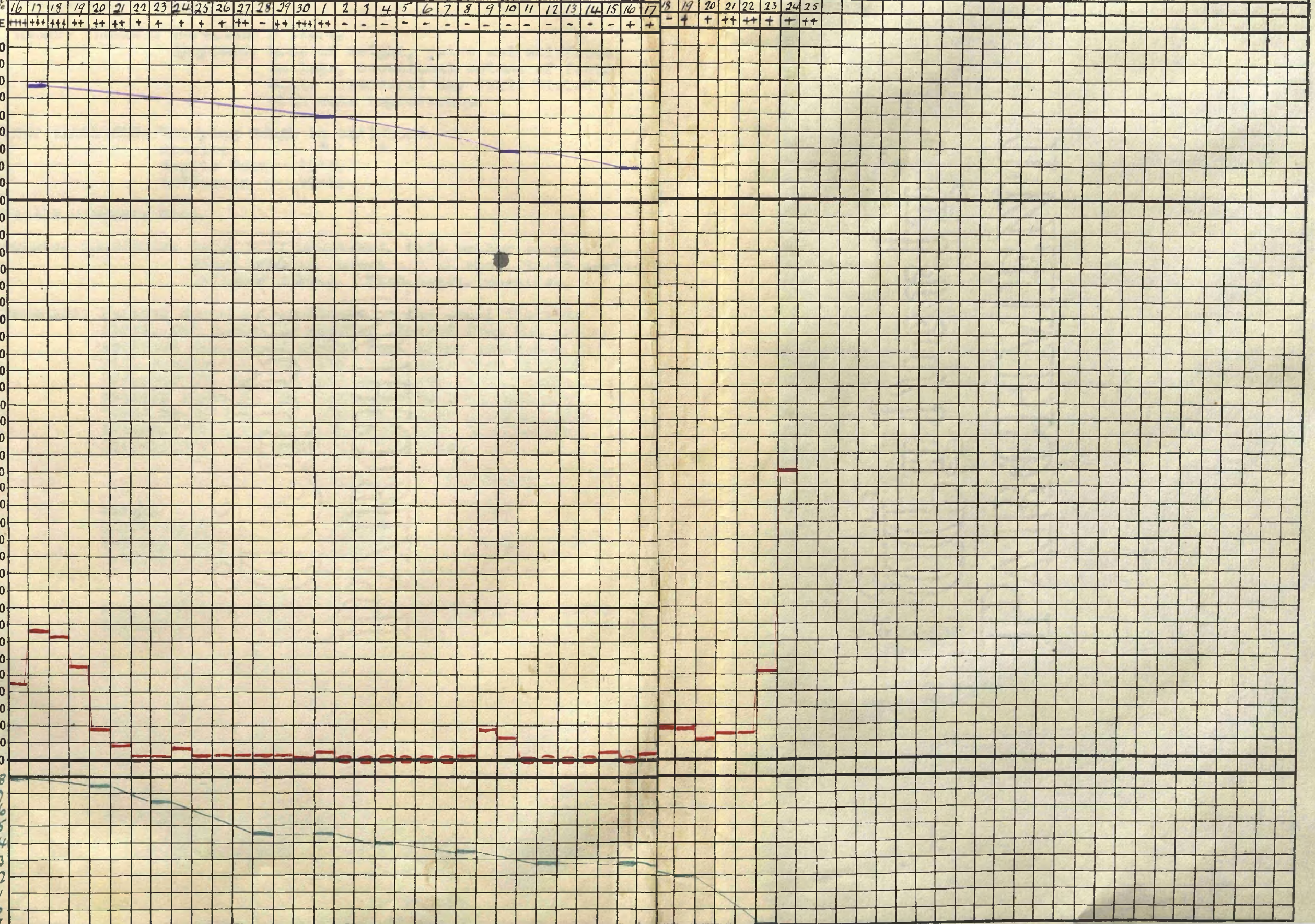
INSULIN UNITS. 0

WEIGHT  
IN KILOS

DIET	GRAMM
1	100
2	200
3	300
4	400
5	500
6	600
7	700
8	800
9	900
10	1000
11	1100
12	1200
13	1300
14	1400
15	1500
16	1600
17	1700
18	1800
19	1900
20	2000
21	2100
22	2200
23	2300
24	2400
25	2500
26	2600
27	2700
28	2800
29	2900
30	3000
31	3100
32	3200
33	3300
34	3400
35	3500
36	3600
37	3700
38	3800
39	3900
40	4000
41	4100
42	4200
43	4300
44	4400
45	4500
46	4600
47	4700
48	4800
49	4900
50	5000
51	5100
52	5200
53	5300
54	5400
55	5500
56	5600
57	5700
58	5800
59	5900
60	6000
61	6100
62	6200
63	6300
64	6400
65	6500
66	6600
67	6700
68	6800
69	6900
70	7000
71	7100
72	7200
73	7300
74	7400
75	7500
76	7600
77	7700
78	7800
79	7900
80	8000
81	8100
82	8200
83	8300
84	8400
85	8500
86	8600
87	8700
88	8800
89	8900
90	9000
91	9100
92	9200
93	9300
94	9400
95	9500
96	9600
97	9700
98	9800
99	9900
100	10000

C.	P.	F.	F.	CALORIES
60	25	5	385	676
30	10	0	160	"
10	0	0	40	"
0	0	0	—	"
0	0	0	—	"
10	0	0	40	"
0	0	0	—	676
0	0	0	—	"
0	0	0	—	"
10	0	0	40	"
20	0	0	80	"
25	10	0	100	"
25	15	0	120	"
30	10	0	160	"
30	10	0	160	"
0	0	0	—	"
15	10	0	100	"
20	15	5	185	"
30	15	5	225	"
40	15	5	265	"
50	20	5	325	"
60	20	10	410	"
0	0	0	—	"
10	15	5	145	"
20	15	5	185	"
25	30	15	355	"
"	"	"	355	"
20	50	30	550	"
0	0	0	—	"
20	60	45	725	"

25	70	50	830
10	0	0	40
20	70	75	895
25	80	85	1185
30	80	95	1295
50	85	100	1440.
"	"	"	"
"	"	"	"





D. T.

Age 22

Grocer's Assistant.

ADMITTED: 21:10:22.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Loss of weight, pains and stiffness  
in legs, occasional oedema of feet.  
Sugar discovered May 1922: dieted  
with some improvement.

PAST ILLNESSES: Whooping Cough in childhood.

Measles " "

Septic Foot 1918.

Influenza 1918.

FAMILY HISTORY: Good.

GENERAL CONDITION: Tall: well nourished. Skin rather rough.

Heart normal. Lungs - ? Rt. apex. Teeth septic

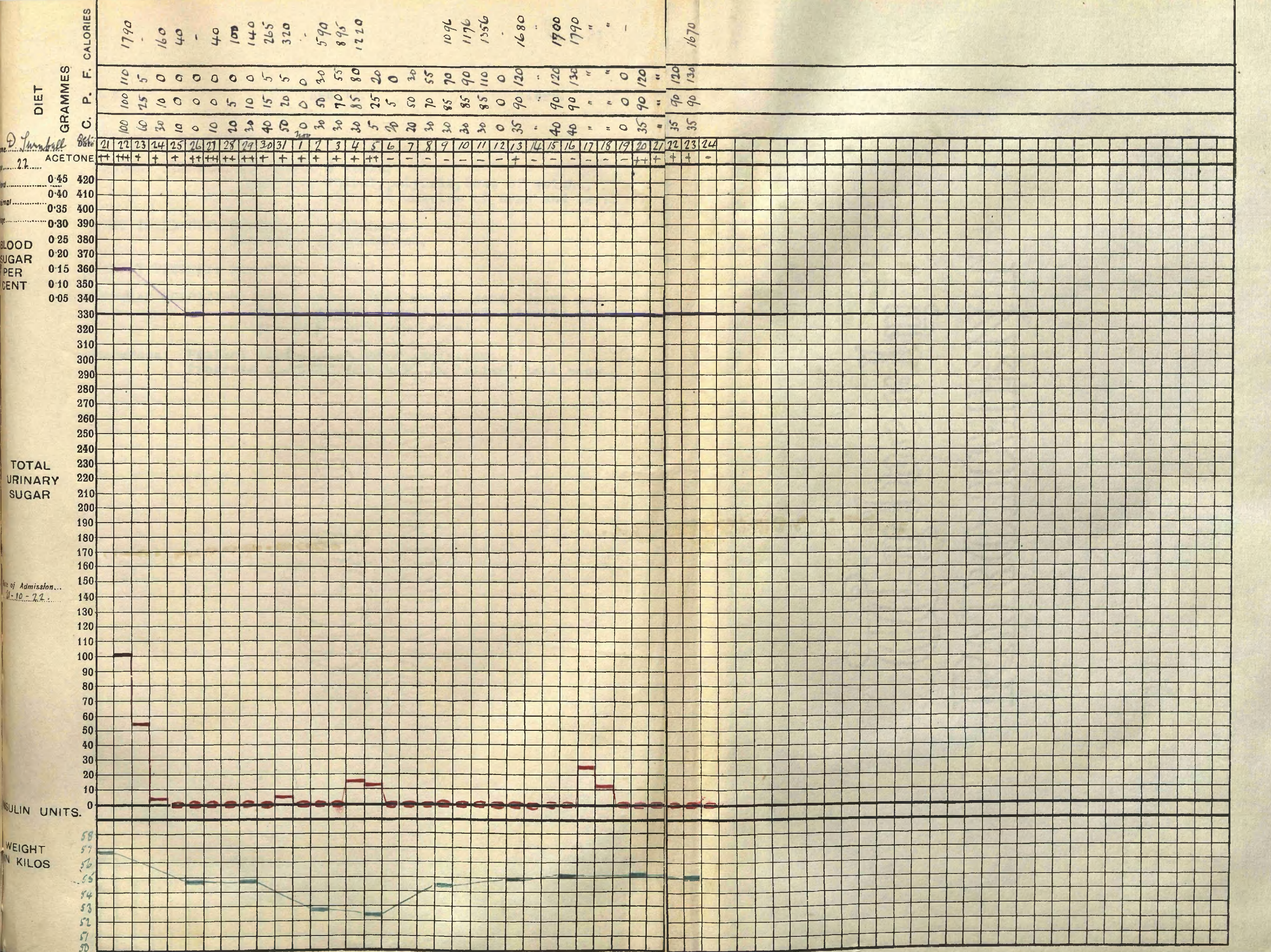
Reflexes normal. Wass. React. negative.

PROGRESS: Patient commenced treatment on the usual dietetic  
lines, and sugar was rapidly cleared from the urine.  
Acidosis was fairly marked just after admission and  
with starvation, but with increasing diet it also  
disappeared. The pain and stiffness in the legs  
rapidly improved. The carbohydrate tolerance was  
found to be low, any increase beyond 35 gms. being  
followed by glycosuria. Patient left hospital on  
35:90:130 = 1670, free from symptoms.

	Admission	Discharge
Weight	56.7k.	55k.
Blood Sugar	0.16%	-
Urinary Sugar	102gms.	Free
Urinary Acetone	++	-

A satisfactory result in a case of moderately severe  
diabetes.







W. J.

Age 17.

Clerk.

ADMITTED: 22:11:22.

PRESENT ILLNESS: Duration: 6 weeks.

Symptoms: Thirst, polyuria, loss of weight,  
pains in muscles of arms and legs.

PAST ILLNESSES: Measles in childhood.

Pneumonia in childhood.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Tall: spare. Skin moist. Acne. Heart and  
lungs normal. Tongue coated. Tonsils slightly  
enlarged. Reflexes normal.

Progress: Patient died 2 days after admission.

Progress has been described in 'Allen' text, page

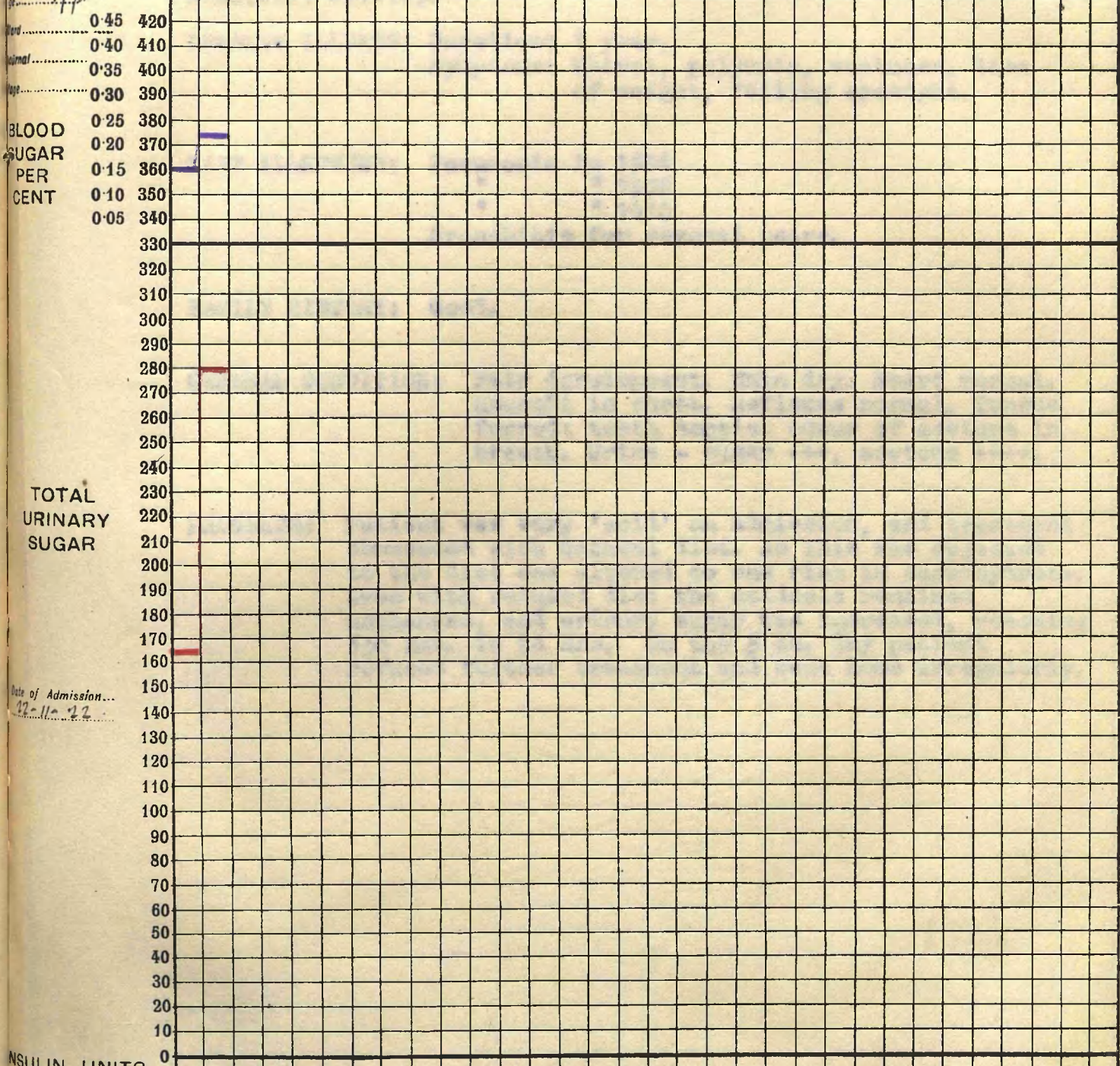


DIET GRAMMES C. P. F. CALORIES

30 36 50

Ordinary 1201 B.

17.7% ACETONE



INSULIN UNITS.

WEIGHT IN KILOS

47

46

45

44

43

42

41

40

39



D.D.

Age 29

Miner.

ADMITTED: 29:1:23.

PRESENT ILLNESS: Duration: 1 year.

Symptoms: Thirst, polyuria, weakness, loss  
of weight, failing eyesight.

PAST ILLNESSES: Pneumonia in 1904

" " 1908

" " 1910

Bronchitis for several years.

FAMILY HISTORY: Good.

GENERAL CONDITION: Fair development. Skin dry. Heart normal.  
Rhonchi in chest. Reflexes normal. Tongue  
furred; teeth septic; odour of acetone in  
breath. Urine - sugar +++, acetone +++++.PROGRESS: Patient was very 'acid' on admission, and treatment  
commenced with oatmeal diet. As this was objected  
to the diet was altered to one rich in carbohydrate.  
Even with reduced diet the acidosis remained  
unchanged, and urinary sugar was increased, reaching  
330 gms. in 24 hrs. On the 5 th. day patient  
refused further treatment and went home irregularly.







Mrs. R.

Age 65.

Housewife.

ADMITTED: 14:4:20.

PRESENT ILLNESS: Duration: Several years.

Symptoms: Thirst, pruritis vulvae, polyuria.

Patient was in hospital with diabetes  
in August 1915.

PAST ILLNESSES: None to previous admission with diabetes.

FAMILY HISTORY: Husband died of diabetes.

GENERAL CONDITION: Stout. Varicose Veins both legs with  
scars of old ulcers. Heart sounds poor:  
pulse irregular (E.S.) Some rale at both  
bases. Tongue furred. Reflexes normal.  
Wass. React. negative.

PROGRESS: Glycosuria was moderate in amount with light diet  
but it quickly cleared up with moderately  
restricted feeding. Patient left hospital on  
35: 55: 150 = 1710, considerably improved. The  
diet furnished as much as she was able to eat.

Weight  
Urinary Sugar  
Urinary Acetone

Admission.

Discharge.

75.5k.  
62 gms.  
-

75k.  
Free.  
-

A case of "Anno Domini" with glycosuria.



Robertson

DIET

GRAMMES

C.	P.	F.	CALORIES
30	40	100	1140.
30	40	120	1385.
30	45	"	1425.
30	"	"	"
30	"	"	1200.
30	"	125	1425.
35	55	150	1710.
4	"	"	"

Date 17 18 19 20 21 22 23 24 25 26 27 28 29

Age 68  
Ward Y1  
Journal 44  
Page 48  
ACETONE  
0.45 420  
0.40 410  
0.35 400  
0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

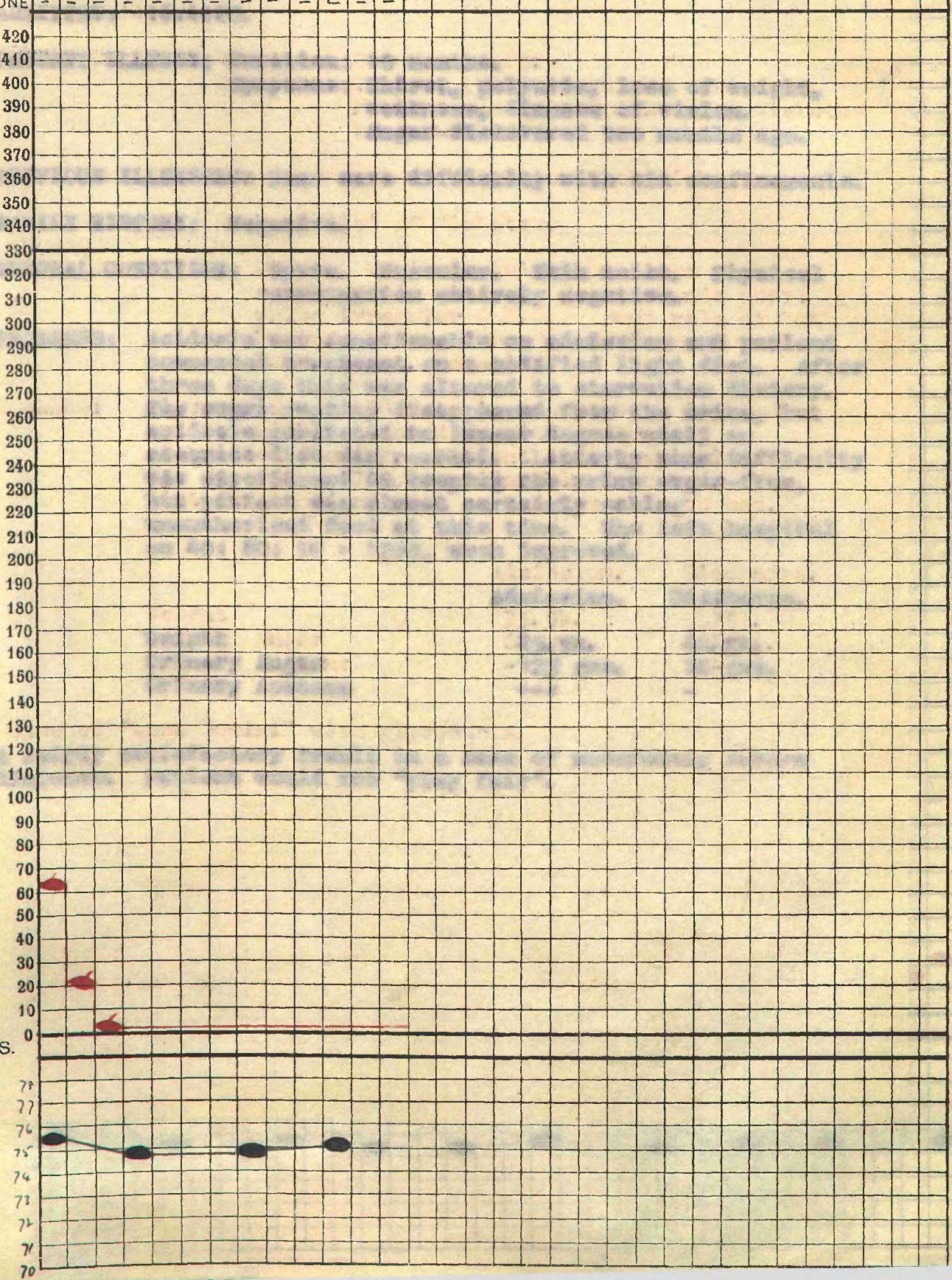
BLOOD  
SUGAR  
PER  
CENT

TOTAL  
URINARY  
SUGAR

Date of Admission...  
14/9/20.

INSULIN UNITS.

WEIGHT  
IN KILOS





Mrs. McG.

Age 29.

Housewife.

ADMITTED: 16:4:20.

PRESENT ILLNESS: Duration: 10 months.

Symptoms: Thirst, polyuria, loss of weight,  
weakness, dimness of vision.

Sugar discovered two months ago.

PREVIOUS ILLNESSES: None save difficulty with six confinements.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Spare. Muscular. Skin moist. Physical  
examination entirely negative.

PROGRESS: Acidosis was considerable on admission and patient commenced treatment on a modified light diet. After three days this was altered to starvation dietary. The sugar rapidly disappeared from the urine, but acidosis persisted in lesser degree until an adequate diet was reached. Latterly some difficulty was experienced in keeping the urine sugar-free, but patient was almost certainly eating unauthorised food at this time. She left hospital on 40: 80: 90 = 1290, much improved.

	Admission.	Discharge.
Weight	45.5k.	44.5k.
Urinary Sugar	125 gms.	10 gms.
Urinary Acetone	+++	-

A fairly satisfactory result in a case of moderately severe diabetes. Patient would not "play fair".



C.G.

Age 21.

Housewife.  
Shop Assistant.

ADMITTED 5:7:20.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Weakness, swelling of the feet, shortness of breath on exertion. Sugar discovered on routine examination in hospital.

PAST ILLNESSES: Whooping Cough in childhood.

Chicken Pox " "

Measles " "

Acute Rheumatism 1904.

Scarlet Fever 1905.

Acute Rheumatism 1911.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Strongly built: well nourished. Thyroid enlarged. K.J.s + . Physical examination

otherwise negative

PROGRESS: Patient showed slight glycosuria on admission ( 0.17% ) but she was placed at once on Ordinary Diet. The urinary sugar cleared up at once, and did not return during her residence. The only restriction in diet was the limiting of white bread to 2 slices per day.

A Glucose Test gave the following result:

Blood Sugar				Urinary Sugar
Before Glucose ( 100 gms. )	0.10%			Free
After " 1/2 hour	0.20%			
" " 1 "	0.15%			Free
" " 1 1/2 "	0.11%			
" " 2 "	0.09%			Free

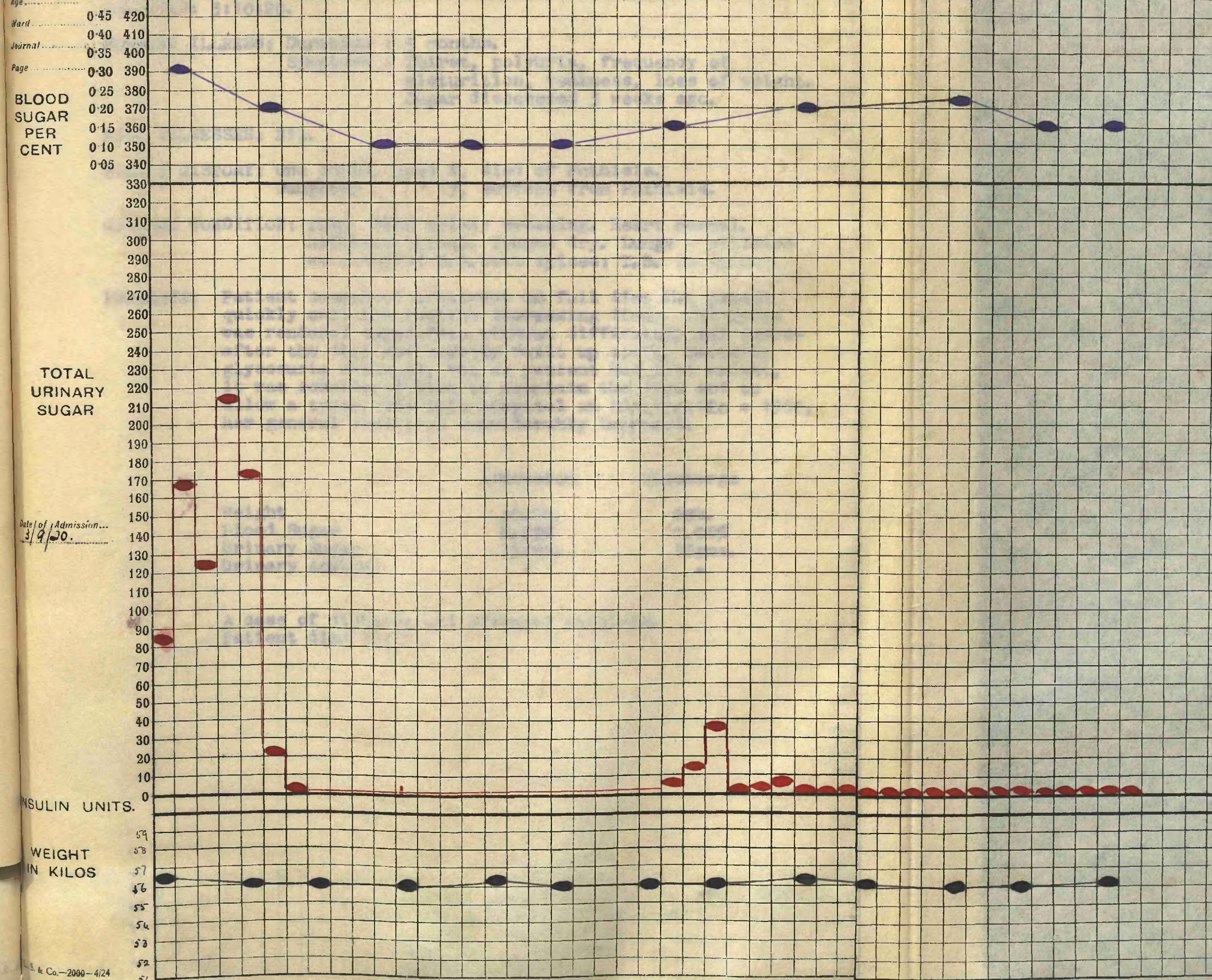
A 'Lag' Curve.

Patient left hospital on full Ordinary Diet free from symptoms.



DIET	GRAMMES	P. F. CALORIES
Supp. Diet.	100 110 112 1848.	
"	80 95 50 1150.	
"	63 54 14 606.	
"	13 6 2 94.	
Full Water Diet.	" " " " " "	
"	13 6 2 94.	
"	26 12 4 188.	
"	39 18 6 282.	
"	39 30 25 501.	
"	39 50 50 806.	
"	40 60 60 940.	
"	50 70 70 1110.	
"	50 85 85 1305.	
"	60 95 95 1475.	
"	60 105 100 1625.	
"	75 " " 1665.	
Vegetable Day.	60 105 105 1665.	
"	60 100 90 1450.	
"	50 85 85 1305.	
"	60 90 90 1450.	
"	100 100 100 1640.	
"	110 110 110 1670.	
"	120 110 110 1750.	
"	130 130 130 1970.	
"	140 140 140 1970.	
"	150 150 150 1970.	
"	160 160 160 1970.	
Vegetable Day.	170 170 170 1970.	

Name *Mr. Brown* Date *3/9/20.*





Mrs J.

Age 51

Housewife.

ADMITTED: 3:10:20.

PRESENT ILLNESS: Duration : 5 months.

Symptoms : Thirst, polyuria, frequency of  
micturition, weakness, loss of weight.  
Sugar discovered 3 weeks ago.

PAST ILLNESSES: Nil.

FAMILY HISTORY: One child, aged 8, died of Phthisis.

Daughter , " 17, suffers from Phthisis.

GENERAL CONDITION: Thin. Skin moist: sweating. Heart normal.  
Reflexes normal. Tongue dry. Lungs - evidence  
well-marked T.B. both apices: T.B. in sputum.

PROGRESS: Patient commenced treatment on full diet but passed  
quickly onto the routine decreasing diets. The urine  
was rendered sugar-free without difficulty, and there-  
after the diet was rapidly built up again. Latterly  
glycosuria returned, but as patient had lost weight,  
it was considered wise to maintain the diet and to  
allow a trace. She left hospital on 100:120:120 = 1960,  
her general condition considerably improved.

	Admission	Discharge
Weight	50.2k.	48k.
Blood Sugar	8.55%	0.05%
Urinary Sugar	75gms.	12gms.
Urinary Acetone	-	-

A case of diabetes and advanced phthisis.  
Patient died 3:3:21.

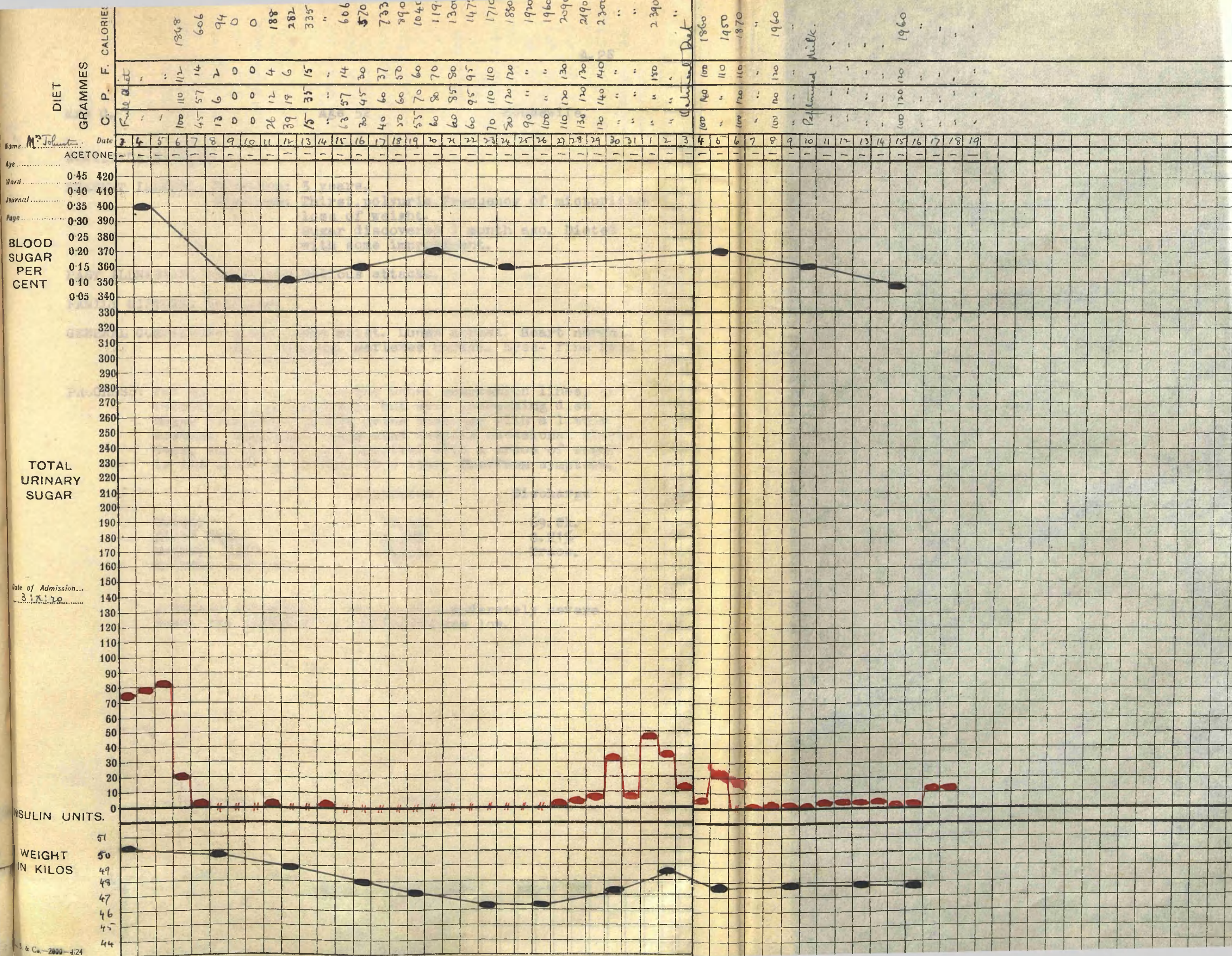


Date of Admission... 3:17:20

INSULIN UNITS.

WEIGHT	50
IN KILOS	49

J. & Co. - 2000 - 4,24





Mrs C.

Age 52

Housewife.

ADMITTED: 8:10:20.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Thirst, polyuria, frequency of micturition  
loss of weight.Sugar discovered 1 month ago. Dieted  
with some improvement.

PAST ILLNESSES: Occasional bilious attacks.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Skin moist. Lungs normal. Heart normal:  
B.P. 140mm.Hg. Reflexes normal. Eyes- fine haze  
in vitreous.PROGRESS: Patient was treated on the usual starvation lines, and  
responded satisfactorily: but with increasing diet  
sugar reappeared in the urine together with a little  
acetone. Glycosuria persisted despite occasional starve  
days, and patient left hospital with a trace of sugar  
in the urine on 40:110:120 = 1590, free from symptoms.

	Admission	Discharge
Weight	70.6k.	69.8k.
Blood Sugar	0.32%	0.21%
Urinary Sugar	50gms.	Trace.
Urinary Acetone	-	-

A fairly satisfactory result in a moderately severe  
case. The carbohydrate tolerance was low.



Mrs. Carson.

Age 34

Ward.....

Journal.....

Page.....

BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

Date of Admission... 8/10/20

INSULIN UNITS.

WEIGHT IN KILOS

DIET

GRAMMES

C. P. F. CALORIES

ACETONE

0.45 420

0.40 410

0.35 400

0.30 390

0.25 380

0.20 370

0.15 360

0.10 350

0.05 340

330

320

310

300

290

280

270

260

250

240

230

220

210

200

190

180

170

160

150

140

130

120

110

100

90

80

70

60

50

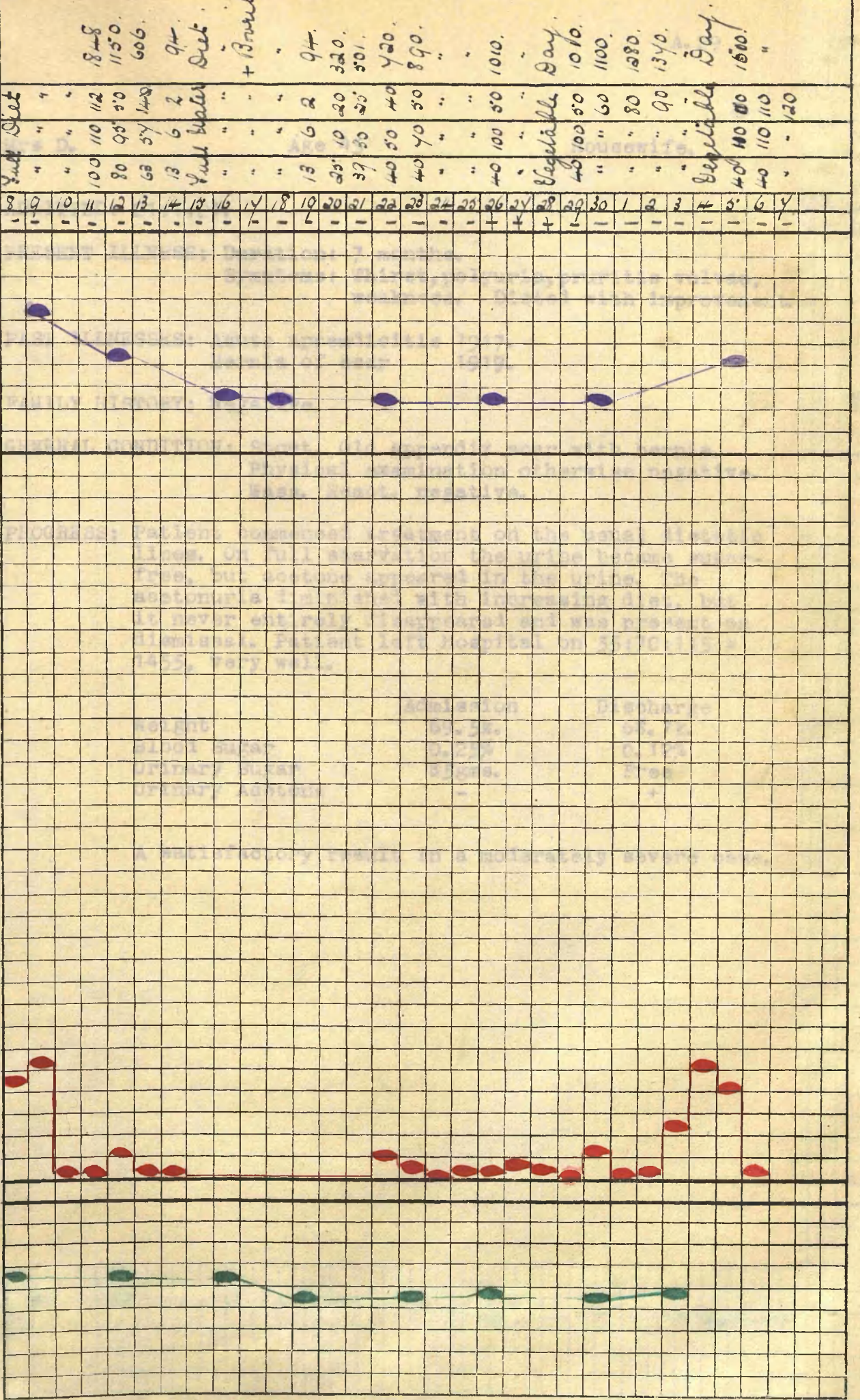
40

30

20

10

0





Mrs D.

Age 45

Housewife.

ADMITTED: 21:1:21.

PRESENT ILLNESS: Duration: 7 months.

Symptoms: Thirst, polyuria, pruritis vulvae,  
weakness. Dieted with improvement.

PAST ILLNESSES: Acute Appendicitis 1917.

Hernia of scar 1919.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Old appendix scar with hernia.

Physical examination otherwise negative.

Wass. React. negative.

PROGRESS: Patient commenced treatment on the usual dietetic lines. On full starvation the urine became sugar-free, but acetone appeared in the urine. The acetonuria diminished with increasing diet, but it never entirely disappeared and was present on dismissal. Patient left hospital on 35:70:115 = 1455, very well.

	Admission	Discharge
Weight	69.5k.	68.7k.
Blood Sugar	0.25%	0.12%
Urinary Sugar	85gms.	Free
Urinary Acetone	-	+

A satisfactory result in a moderately severe case.



Name *Mrs. Dalgligh* Date *21*  
 Age *6* ACETONE *-*  
 Ward *44*  
 Journal *44*  
 Page *0.30*

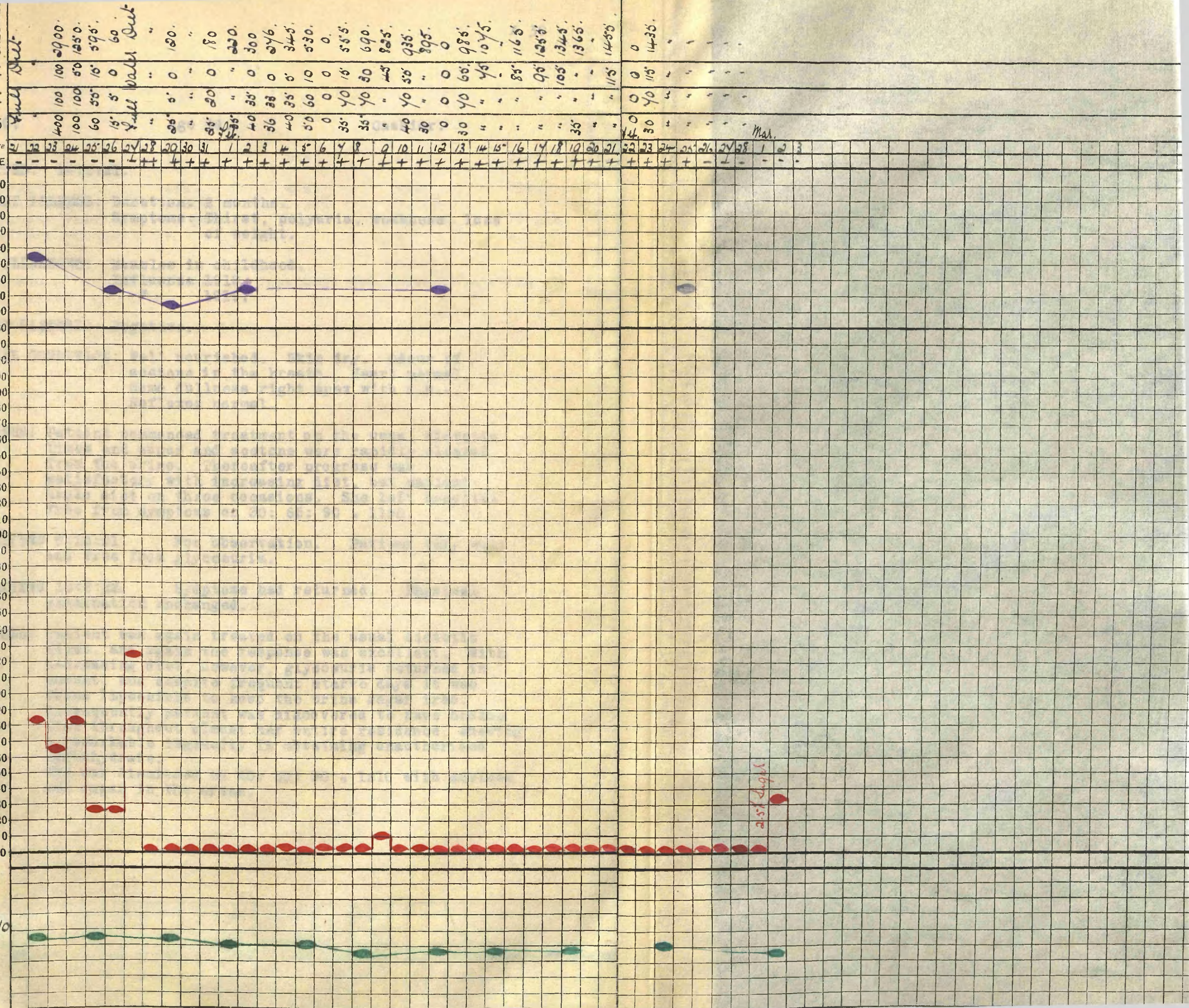
BLOOD SUGAR PER CENT  
 0.25 380  
 0.20 370  
 0.15 360  
 0.10 350  
 0.05 340

TOTAL URINARY SUGAR

Date of Admission... *21/1/21*

INSULIN UNITS.

WEIGHT IN KILOS  
*40*





M. McC.

Age 14.

Cashier.

ADMITTED: 14:2:21.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Thirst, polyuria, weakness, loss of weight.

PAST ILLNESSES: Measles in childhood.

Influenza 1918. Treatment was impossible owing to patient's habits. 1919.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished. Skin dry. Odour of acetone in the breath. Heart normal. Some dullness right apex with R.M.--. Reflexes normal.

PROGRESS: Patient commenced treatment on the usual dietetic lines and sugar and acetone were rapidly cleared from the urine. Thereafter progress was satisfactory with increasing diet, but patient broke diet on three occasions. She left hospital free from symptoms on 30: 65: 90 = 1190.

READMITTED 8:10:21. For observation. Patient very well and free from glycosuria.

READMITTED 15:2:22. Symptoms had returned. Physical examination unchanged.

PROGRESS: Patient was again treated on the usual dietetic lines, and again the response was excellent. With increasing diet, however, glycosuria returned in amount, and despite frequent starve days it was found impossible to keep the urine sugar free. Subsequently patient was discovered to have broken diet throughout almost her entire residence, shewing a remarkable ingenuity in obtaining unauthorised carbohydrate. She was dismissed on 40: 60: 90 = 1210 with acetone and sugar in the urine.







## GRAMMES

C. P. F. CALORIES

name *W. Ballum* Date \_\_\_\_\_  
ACETONE

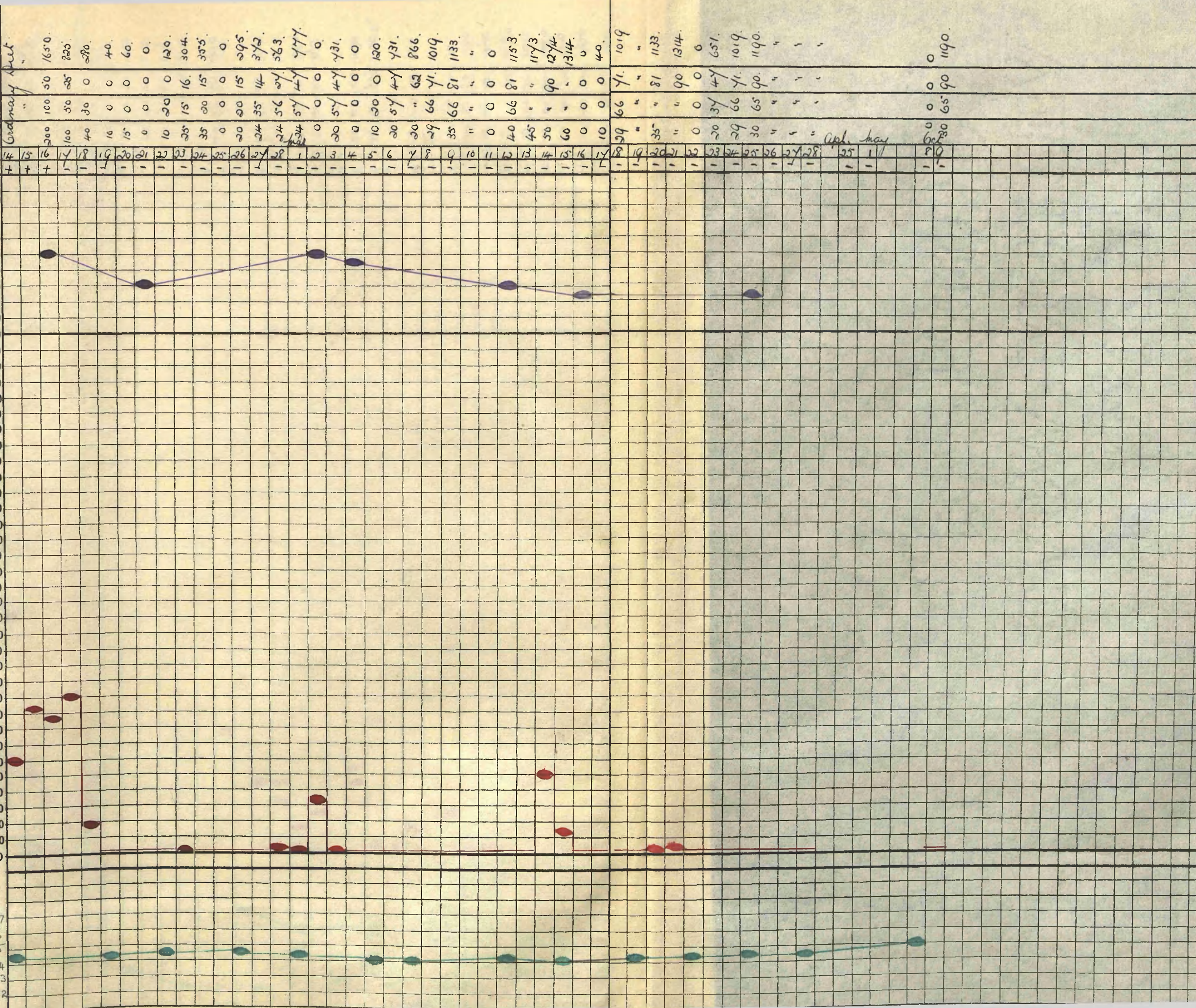
Age	0.45	420
Ward	0.40	410
Journal	0.35	400
Page	0.30	390
BLOOD	0.25	380
SUGAR	0.20	370
PER	0.15	360
CENT	0.10	350
	0.05	340

TOTAL	230
URINARY	220
SUGAR	210

Date of Admission... 150  
14/2/21. 140

INSULIN UNITS.

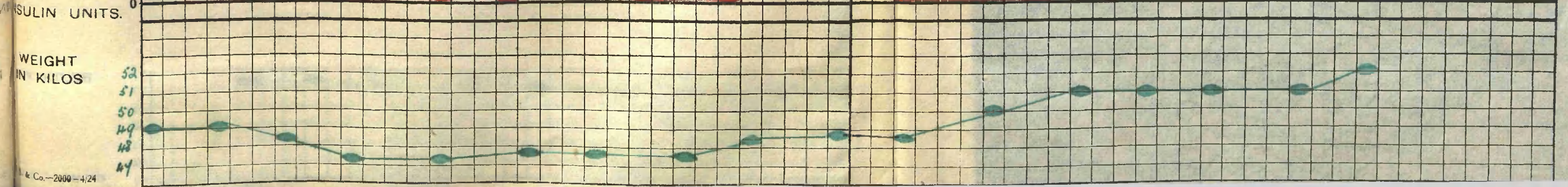
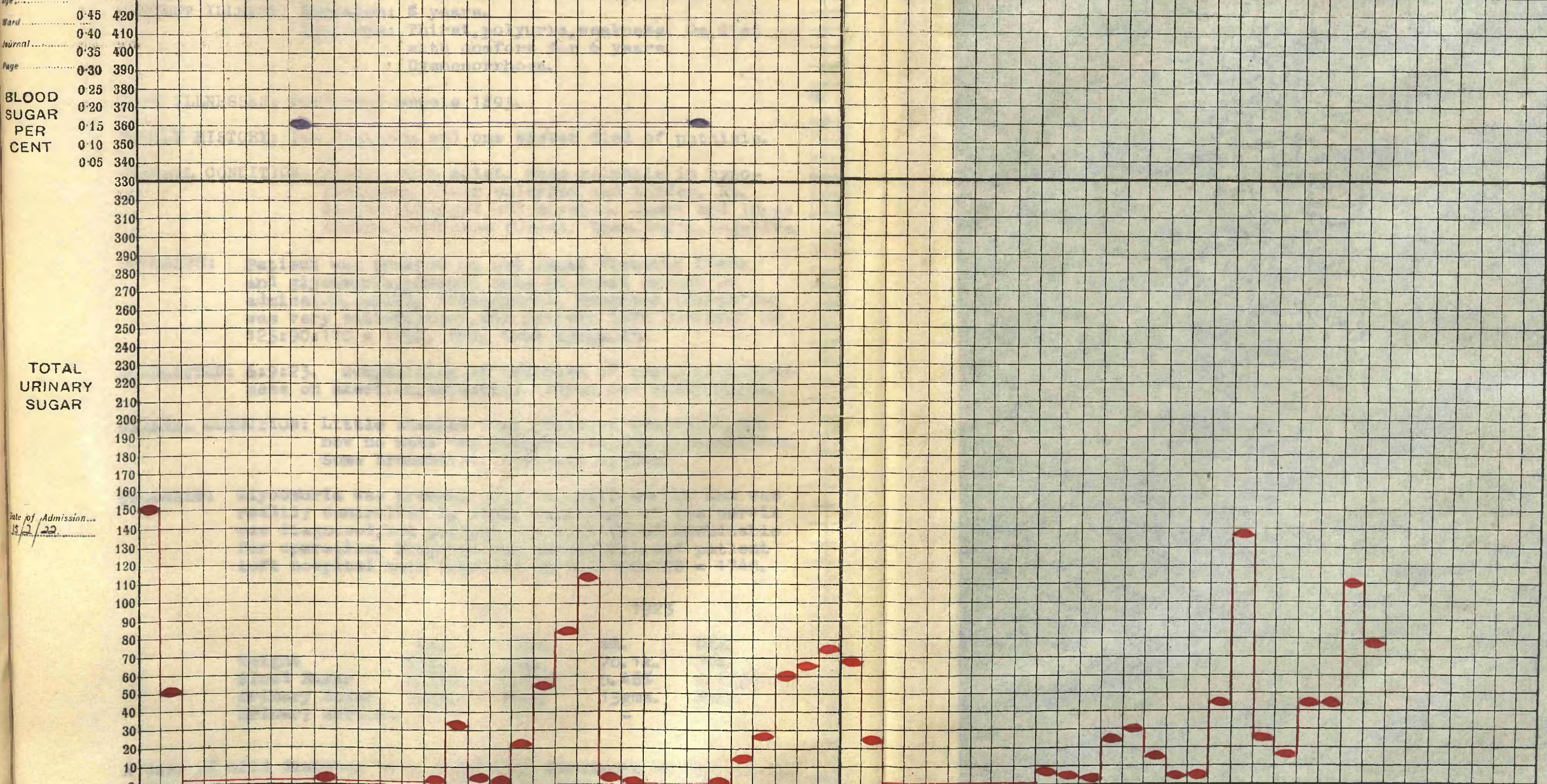
WEIGHT  
IN KILOS





Name	Date	DIET		C.	P.	F.	CALORIES
		Light	Dark				
Mary McCallum	15	60	25	385			
	16	30	10	160			
	17	10	0	40			
	18	"	"	"			
	19	20	10	210			
	20	30	20	420			
	21	40	20	500			
	22	50	30	640			
	23	60	55	865			
	24	0	0	0			
	25	40	60	1030			
	26	"	"	"			
	27	40	70	1250			
	28	20	100	1385			
	29	30	10	120			
	30	40	20	460			
	31	20	10	120			
	32	40	60	1030			
	33	"	"	"			
	34	20	40	500			
	35	0	0	0			
	36	10	30	340			
	37	15	40	400			
	38	20	50	550			
	39	20	60	680			
	40	20	60	860			
	41	"	"	950			
	42	30	70	1120			
	43	"	"	"			
	44	30	80	1120			
	45	10	30	390			
	46	"	"	"			
	47	15	40	630			
	48	15	50	760			
	49	20	60	910			
	50	25	80	1060			
	51	30	80	1080			
	52	"	"	1140			
	53	10	30	390			
	54	25	60	1060			
	55	"	"	"			
	56	0	0	0			
	57	60	90	1150			
	58	40	100	1180			
	59	"	"	"			
	60	20	30	390			
	61	60	80	1060			
	62	"	"	"			
	63	55	45	865			
	64	"	"	"			
	65	60	90	1210			

Age	ACETONE	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98	99	100
-----	---------	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	-----





Mrs C.

Age 47

Housewife.

ADMITTED: 12:3:21.

PRESENT ILLNESS: Duration: 8 years.

Symptoms: Thirst, polyuria, weakness. On diet with comfort for 6 years. Dysmenorrhoea.

PAST ILLNESSES: Puerperal sepsis 1895.

FAMILY HISTORY: Two brothers and one sister died of phthisis.

GENERAL CONDITION: Stout: skin moist. Mass palpable in hypogastrium. Liver enlarged and tender. Rt. kidney enlarged and movable. Heart and lungs normal. Reflexes normal. Wass. React. negative.

PROGRESS: Patient was treated on the usual dietetic lines, and glycosuria, present only in small amount on admission, quickly disappeared. Progress thereafter was very satisfactory, and patient left hospital on 125:90:110 = 1850, free from symptoms.

READMITTED: 6:9:23. Complaining of weakness of legs, breathlessness on exertion, defective vision, and menorrhagia.

GENERAL CONDITION: Little changed from previous admission, but now no mass was palpable in the hypogastrium. Some bronchitis. Reflexes normal.

PROGRESS: Glycosuria was present only in small amount and was readily controlled by diet. Carcinoma of the cervix was diagnosed, but patient was considered unsuitable for operation. Progress was uneventful, and patient left hospital much improved on 100:100:120 = 1740.

1921

1923

	Ad.	Dis.	Ad.	Dis.
Weight	63k.	58k.	70.1k.	70k.
Blood Sugar	0.11%	0.21%	0.46%	0.175%
Urinary Sugar	5gms.	Free	15gms.	Free.
Urinary Acetone	-	-	-	-

A case of mild diabetes in a woman with Carcinoma of the cervix.



Name Mr. Cochran Date 12/3/21

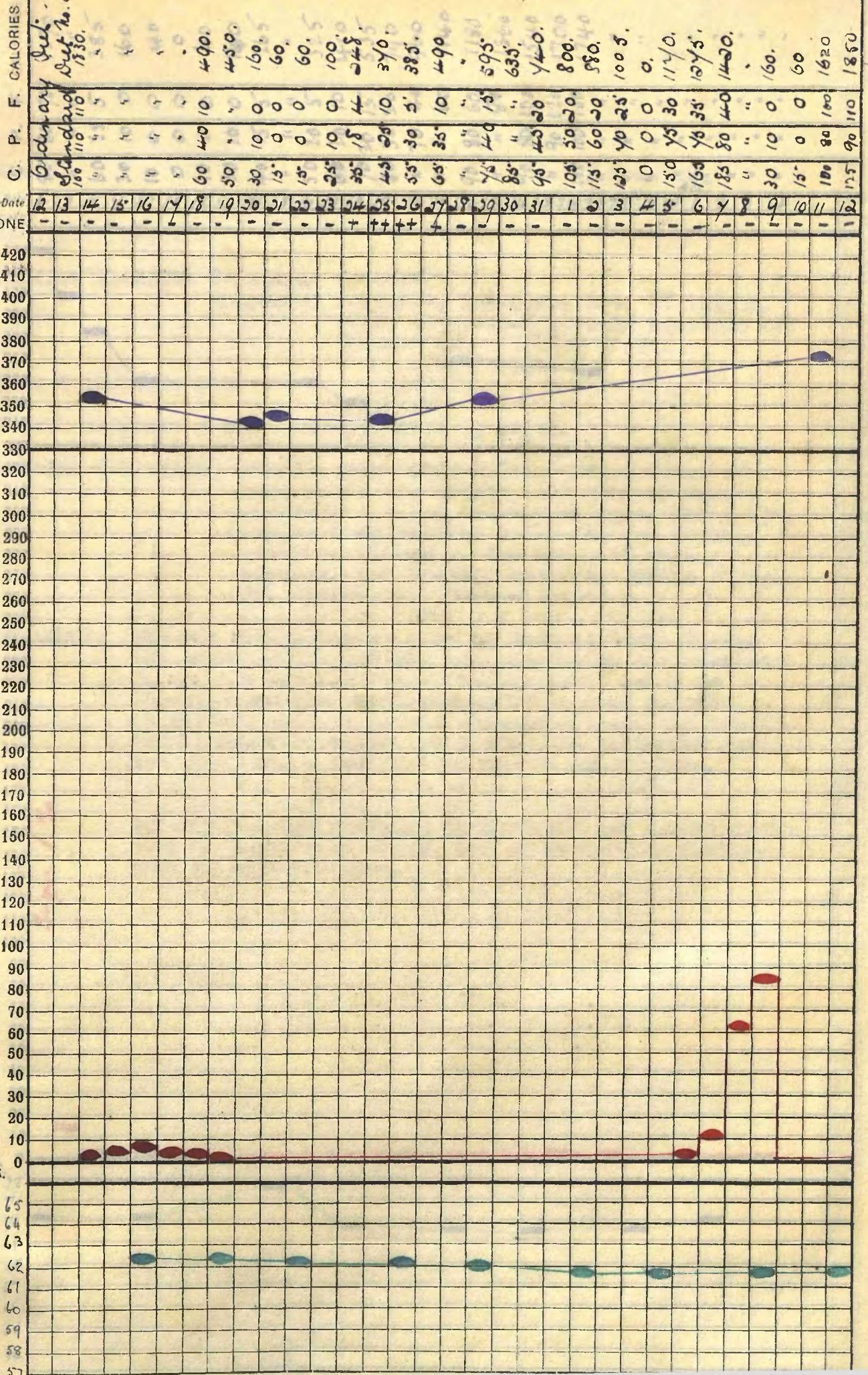
Age 52  
Ward 420  
Journal 410  
Page 400  
BLOOD SUGAR PER CENT  
0.45 380  
0.40 370  
0.35 360  
0.30 350  
0.25 340  
0.20 330  
0.15 320  
0.10 310  
0.05 300

TOTAL URINARY SUGAR

Date of Admission... 12/3/21

INSULIN UNITS.

WEIGHT IN KILOS









Mrs R.

Age 28

Housewife.

ADMITTED: 28:5:21.

PRESENT ILLNESS: Duration: 6 weeks.

Symptoms: Polyuria, weakness, loss of weight.

Sugar discovered 5 weeks ago: put off  
'sweets'.

PAST ILLNESSES: Nil.

FAMILY HISTORY: 3 last children died at birth.

GENERAL CONDITION: Small: thin. Skin moist. Marked odour of acetone in the breath. Air hunger. Heart slightly + : V.S. murmur at apex. Pulse 130. Lungs- respirations noisy: 40 per minute. Liver +. Spleen +. She was quite clear mentally but readily excited. Urine - sugar 2%. Acetone + . Diacetic Acid - .

PROGRESS: Patient was placed at once on Oatmeal diet. During the night of 28:5:24 she was very noisy and restless. On 29:5:24 she was rather better, there being no cyanosis and the respirations being much quieter. On 30:5:24 patient became unconscious and the breathing became very noisy. At 3 a.m. she collapsed suddenly and died. There was no Post Mortem examination.

As in this case evidence of acidosis was scanty death must be considered as due rather to Cardiac failure than to Diabetic Coma.



Mrs. McE.

Age 70.

Housewife.

ADMITTED: 12:11:21.

**PRESENT ILLNESS:** Duration: Probably some years.  
Symptoms: Patient was admitted suffering from pains in the back and legs accompanied by headache and vomiting.

**PAST ILLNESSES:** Abscess on chest wall, ? T.B. 1890.

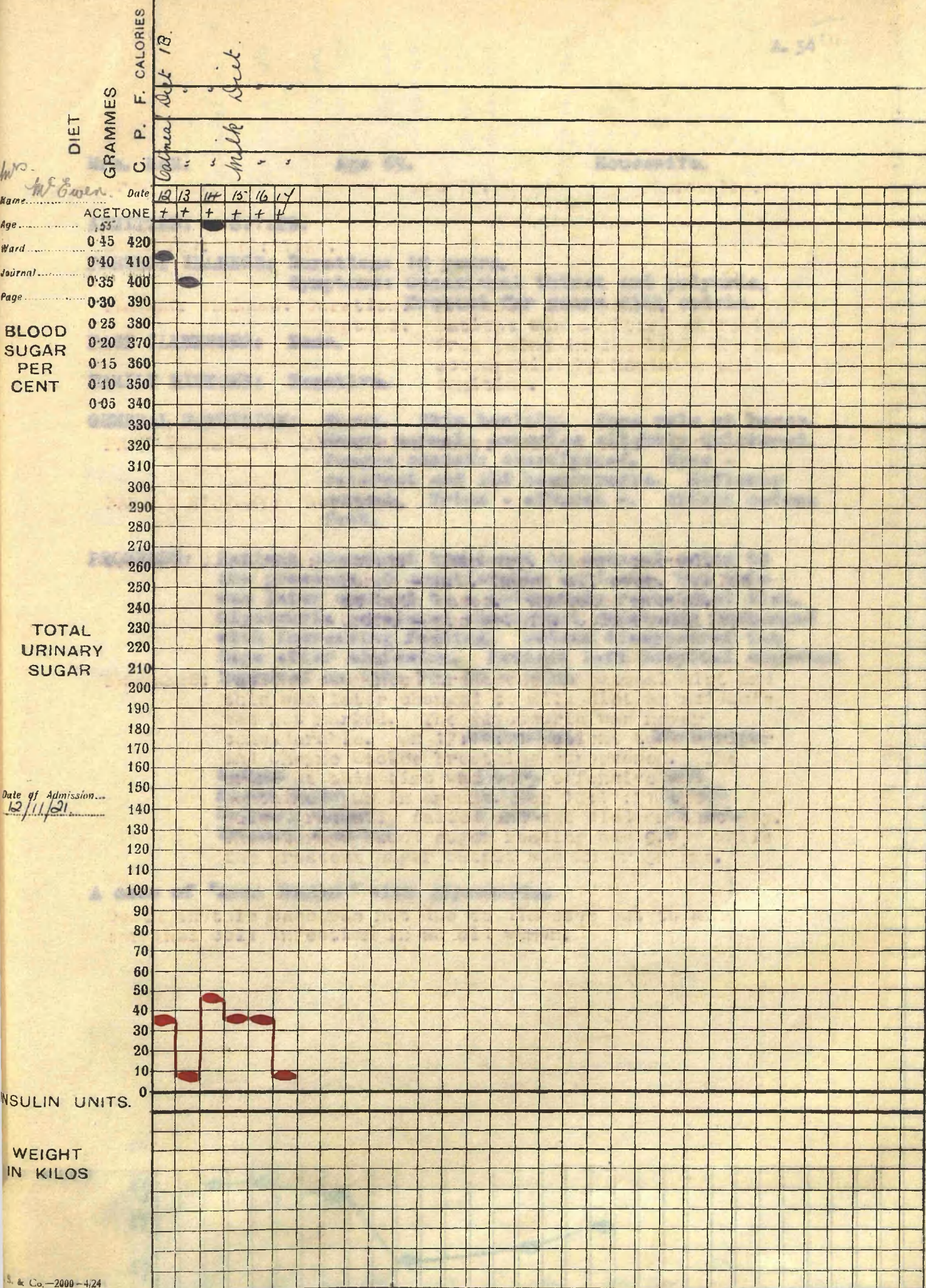
**FAMILY HISTORY:** Negative.

**GENERAL CONDITION:** Stout. Skin dry. Heart normal.  
Lungs - R.M. harsh all over. Rales at both bases. Reflexes normal.  
Urine - pus ++. (B. Coli Pyelitis)

**PROGRESS:** Patient commenced treatment on oatmeal diet and this was later changed to milk diet as acidosis was not marked. The glycosuria was never considerable. On 17:11:21 patient took a rigor and Cheyne Stokes breathing supervened. The urine at this time was very offensive and contained pus in amount. On 18:11:21 the pulse gradually failed and she died on that day. The average blood sugar reading was 9.45% while the greatest sugar output was under 50 gms.

Death in this case was not due to diabetes but to a terminal coli infection in an old woman.







Mrs. McN.

Age 69.

Housewife.

ADMITTED: 6:1:22.

PRESENT ILLNESS: Duration: 10 years.

Symptoms: Occasional thirst and polyuria.  
Treated for years with codeia.

PAST ILLNESSES: None.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Stout. Skin healthy. Some rale at bases.  
Heart normal: arteries slightly thickened.  
Tongue coated: constipated. Eyes -  
cataract and old haemorrhages. Reflexes  
normal. Urine - albumen +. Slight oedema  
feet.

PROGRESS: Patient commenced treatment on oatmeal owing to  
the presence of considerable acidosis, but this  
was later changed to a moderately restricted diet.  
Glycosuria persisted throughout remaining unchanged  
with increasing feeding. Oedema disappeared ten  
days after admission. Patient left hospital somewhat  
improved on 150: 70: 60 = 1420.

	Admission.	Discharge.
Weight	59.2k.	58k.
Blood Sugar	0.15%	0.16%.
Urinary Sugar	8 gms.	12 gms.
Urinary Acetone	+	+

A case of "Anno Domini" with glycosuria.







M.R.

Age 24.

At Home.

ADMITTED 14:1:22.

PRESENT ILLNESS: Duration: 2 years.

Symptoms: Thirst, polyuria, weakness, loss of weight.

Sugar discovered 18 months ago:  
dieted with slight improvement.

PAST ILLNESSES: Scarlet Fever in childhood

Measles " "

Mitral Stenosis 1919: occasional break-downs  
of compensation.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Very emaciated. Marked generalised oedema.

Skin moist. Heart- V.S. murmur all over  
precordium: presystolic and diastolic  
murmurs at apex. Some rale at both bases.  
Liver and spleen both palpable. Reflexes  
normal. Tongue coated: breath smelling  
of acetone. Urine - albumen ++.

PROGRESS: Patient commenced treatment on oatmeal diet. The sugar output was greatly reduced but pari passu the oedema increased enormously and ascites became very marked. With increasing feeding the glycosuria again became very marked but the oedema gradually disappeared. Acetonuria was present from time to time but never, to an alarming extent. By 10:3:22 the oedema had entirely disappeared and the sugar output was under 20 gms. Patient left hospital on 22:3:22 with slight glycosuria and with a diet of 60: 80: 110 = 1550.

## Admission.

## Discharge.

Weight	37k.	37.3k.
Blood Sugar	0.35%	0.25%.
Urinary Sugar	154 gms.	33 gms.
Urinary Acetone	++	+

A case of Mitral Stenosis with diabetes.







Mrs. A.

Age 64.

Housewife.

ADMITTED: 7:3:22.

PRESENT ILLNESS: Duration: Several years.

Symptoms: Thirst, polyuria, weakness, pains  
in right arm, sugar discovered  
December 1921.PAST ILLNESSES: Typhus Fever 1869.  
Typhoid Fever 1873.  
Gall Stones 1906.  
Influenza 1920.

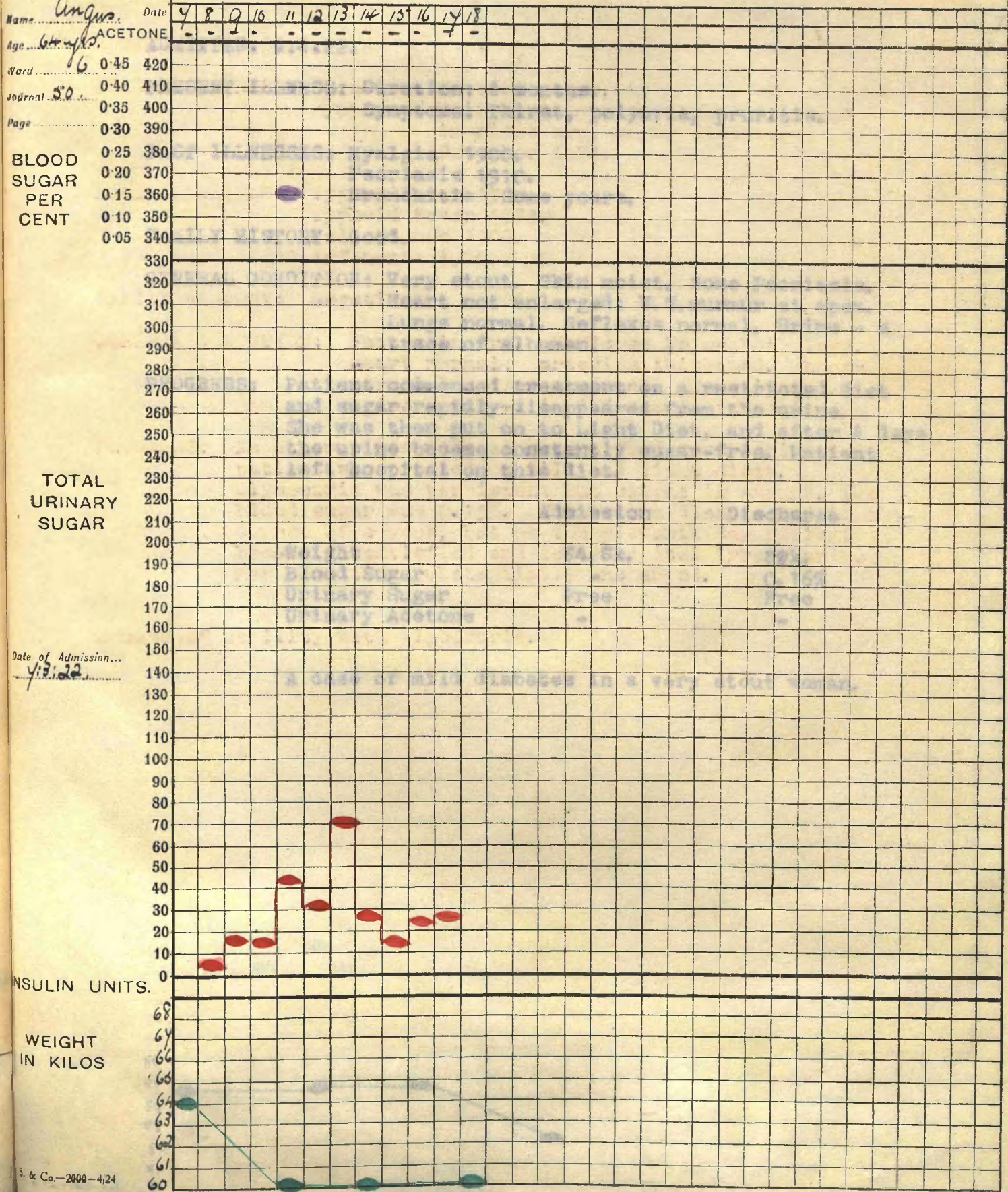
FAMILY HISTORY: Negative.

GENERAL CONDITION: Fat, skin dry. Varicose Veins both legs.  
Heart normal: arteries thickened. Lungs  
normal. Reflexes normal. Constipated.  
Urine - pus +. (Mild B.Coli infection)PROGRESS: No attempt was made at severe food restriction and  
patient was treated on milk and light diets.  
Glycosuria was persistent but slight in amount. The  
blood sugar was 0.15%. The oedema disappeared in the  
course of a week, but on the eleventh day patient  
became dissatisfied and left hospital irregularly,  
her condition substantially unchanged.

A case of senility with glycosuria.



DIET GRAMMES  
C. P. F. CALORIES  
Name: *Angus* Date: *4 8 9 10 11 12 13 14 15 16 17 18*  
Age: *64 yrs.* ACETONE: *- - - - -*  
Ward: *6* 0.45 420  
Journal: *50* 0.40 410  
Page: 0.35 400  
0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340  
330  
320  
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300  
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190  
180  
170  
160  
150  
140  
130  
120  
110  
100  
90  
80  
70  
60  
50  
40  
30  
20  
10  
0  
INSULIN UNITS.  
WEIGHT IN KILOS  
68  
67  
66  
65  
64  
63  
62  
61  
60





Mrs S.

Age 49

Housewife.

ADMITTED: 6:4:22.

PRESENT ILLNESS: Duration: 4 months.

Symptoms: Thirst, polyuria, pruritis.

PAST ILLNESSES: Myalgia 1900.

Psoriasis 1910.

Bronchitis Some years.

FAMILY HISTORY: Good.

GENERAL CONDITION: Very stout. Skin moist. Some Psoriasis.

Heart not enlarged: V.S.murmur at apex.

Lungs normal. Reflexes normal. Urine - a trace of albumen.

PROGRESS: Patient commenced treatment on a restricted diet and sugar rapidly disappeared from the urine. She was then put on to Light Diet, and after 4 days the urine became constantly sugar-free. Patient left hospital on this diet.

Admission

Discharge

Weight

84.8k.

82k.

Blood Sugar

-

0.16%

Urinary Sugar

Free

Free

Urinary Acetone

-

-

A case of mild diabetes in a very stout woman.



Name M. S. Shaw Date 6

Age 7 ACETONE

Ward 0.45 420

Journal 0.40 410

Page 0.35 400

0.30 390

BLOOD 0.25 380

SUGAR 0.20 370

PER 0.15 360

CENT 0.10 350

0.05 340

330

320

310

300

290

280

270

260

250

240

230

220

210

200

190

180

170

160

150

140

130

120

110

100

90

80

70

60

50

40

30

20

10

0

INSULIN UNITS.

WEIGHT IN KILOS

88

87

86

85

84

83

82

81

80

79

78

77

76

75

74

73

72

71

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DIET

GRAMMES

C. P. F. CALORIES

60 25 5 385

30 10 0 160

Light Diet.

38

37

36

35

34

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32

31

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11

10

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1

0

DIET

GRAMMES

C. P. F. CALORIES

60 25 5 385

30 10 0 160

Light Diet.

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DIET

GRAMMES

C. P. F. CALORIES

60 25 5 385

30 10 0 160

Light Diet.

38

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11

10

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DIET

GRAMMES

C. P. F. CALORIES

60 25 5 385

30 10 0 160

Light Diet.

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DIET

GRAMMES

C. P. F. CALORIES

60 25 5 385

30 10 0 160

Light Diet.

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Mrs. D.

Age 52.

Housewife.

ADMITTED: 30:7:22.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Thirst, polyuria, loss of weight,  
pruritis vulvae, sleeplessness.

PAST ILLNESSES: Scarlet Fever in infancy.

Measles " "

Pneumonia 1913.

Hysterectomy 1915.

FAMILY HISTORY: One child died of Hip Joint Disease.

GENERAL CONDITION: Stout. Florid. Skin moist. Scar left  
neck. Arteries thickened. B.P. 225 mm. Hg.  
Lungs normal. Reflexes normal.PROGRESS: Patient was treated on the usual dietetic lines.  
Glycosuria, at no time marked, rapidly cleared up  
and did not again recur. Patient left hospital  
on 120: 90: 120 = 1920, free from symptoms.

	Admission.	Discharge.
Weight	82k.	82k.
Blood Sugar	0.15%	0.15%
Urinary Sugar	12 gms.	Free.
Urinary Acetone	-	-

A case of mild diabetes.







Mrs. S.

Age 52.

Housewife.

ADMITTED: 20:10:22.

PRESENT ILLNESS: Duration: 1 year.

Symptoms: Weakness, loss of weight, profuse sweating, pruritis vulvae.

PAST ILLNESSES: Whooping Cough in childhood.  
Measles in childhood.

FAMILY HISTORY: Six of twelve children died in infancy.

GENERAL CONDITION: Stout. Skin dry. Varicose Veins in both legs. Heart and lungs normal. Tongue dry. Reflexes normal.

PROGRESS: Patient commenced treatment on an ordinary diet and glycosuria was minimal. With reduced carbohydrate a trace of sugar persisted which disappeared on starvation. Patient was dismissed on ordinary diet less obvious sugar with a trace of sugar in the urine.

A case of obesity with glycosuria. Not suffering from diabetes.



Skull Water Diet  
Ord. Diet. (carb.)



Mrs. M.

Age 51.

Housewife.

ADMITTED: 21:10:22.

PRESENT ILLNESS: Duration: 5 years.

Symptoms: Weakness, loss of weight,  
sleeplessness, pruritis vulvae.

PAST ILLNESSES: Scarlet Fever in childhood.

Measles " "

FAMILY HISTORY: Negative.

GENERAL CONDITION: Very fat. Skin healthy. Lungs normal.

Heart - V.S. murmur at apex: second  
aortic + : B.P. 180. mm. Hg. Reflexes  
normal. Tongue coated.PROGRESS: Patient commenced treatment on the usual dietetic  
lines and with decreasing diet the sugar output  
was greatly reduced: but patient refused to submit  
to starvation and left hospital irregularly on  
the fifth day.READMITTED: 7:9:24. Patient had been dieting to some extent  
since previous dismissal, but all her symptoms had  
returned with, in addition, thirst and polyuria.GENERAL CONDITION: Substantially unchanged, but skin now very  
dry with considerable inflammation around the vulva.PROGRESS: Patient again commenced treatment on decreasing diet,  
but she was found to be eating unauthorised carbohydrate  
and was discharged irregularly.Apparently a case of moderately severe diabetes. Treatment was  
impossible owing to patient's misbehaviour.



DIET		GRAMMES		C.		P.		F.		CALORIES	
1790.		110		100		100		110		1790.	
385.		5		35		35		5		385.	
160.		0		10		10		0		160.	
40.		0		0		0		0		40.	

Name	Date	ACETONE
Ward	21	1
Journal	22	1
Page	23	1
	24	1
	25	1

BLOOD SUGAR PER CENT	0.45	0.40	0.35	0.30	0.25	0.20	0.15	0.10	0.05
420									
410									
400									
390									
380									
370									
360									
350									
340									
330									
320									
310									
300									
290									
280									
270									
260									
250									
240									
230									
220									
210									
200									
190									
180									
170									
160									
150									
140									
130									
120									
110									
100									
90									
80									
70									
60									
50									
40									
30									
20									
10									
0									

TOTAL URINARY SUGAR	140	130	120	110	100	90	80	70	60	50	40	30	20	10	0
140															
130															
120															
110															
100															
90															
80															
70															
60															
50															
40															
30															
20															
10															
0															

INSULIN UNITS.	80	70	60	50	40	30	20	10	0
80									
70									
60									
50									
40									
30									
20									
10									
0									

WEIGHT IN KILOS	80	70	60	50	40	30	20	10	0
80									
70									
60									
50									
40									
30									
20									
10									
0									

Date of Admission...  
21/10/22

12.5% sugar



SYNOPSIS OF CASES

INSULIN TREATMENT

'PEDIGREE' SERIES

MALE

P. 1 - P. 5.

FEMALE

P. 9 - P. 14.



Case	Age	Emaciated	Acid	Immed. Insulin		Later Insulin		Weight Kilo.	Carbo. Toler.
				Max.	Dis.	Max.	Dis.		
P. 1	42	+		45	30			-2	+60
P. 2	13	+	+	35	20			+6	+25
P. 3	38	+		65	55			+3	-40
P. 4	17		+			30	25	0	-20
P. 5	34	+	+			20	5	+1	+10
P. 6	31		+	50	30			0	0
P. 7	42		+			35	5	0	+30
P. 8	5					25	25	+3	+30
P. 9	33					25	23	0	-70
P. 10 <sup>1</sup>	50 <sup>1</sup>								
P. 11	62					5	0	-1	+30
P. 12	34	+		50	40			0	+30
P. 13	39					40	0	+1	+50
P. 14 <sup>*</sup>	15					15	5	0	+105

<sup>1</sup> Patient admitted with Haematemesis.

<sup>\*</sup> Patient suffering from Phthisis.



J.C.

Age 42

Plasterer.

ADMITTED: 2:1:20.

PRESENT ILLNESS: Duration: 3 months.

Symptoms: Thirst, polyuria, loss of weight.

Sugar discovered 28:12:19.

PAST ILLNESSES: Indigestion 1919.

Haemorrhoids for 3 years.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin: muscular. Skin dry and slightly  
bronzed. Reflexes normal. Teeth carious.

PROGRESS: Progress was very satisfactory for two months, sugar and acetone giving little trouble. But latterly the tolerance appeared to fall, and glycosuria was constantly present on a lower diet, though never in large amount. He left hospital on 40:80:175 = 2055, with a trace of sugar in the urine.

READMITTED: 28:5:21. Patient had been reporting each month since dismissal on 1:4:20, and had been very well and constantly at work. Latterly, however, he had noted an increase in the glycosuria in spite of numerous starve days. As he shewed 16% sugar in the urine he was admitted for treatment. The general condition was unchanged save that the K.J.s could not now be elicited.

PROGRESS: Patient responded very well to dietetic treatment, acetone and sugar being rapidly cleared from the urine. He left hospital on 40:80:110 = 1470, free from symptoms.

READMITTED: 28:11:22. Patient had been fairly well since last admission, but latterly there had been a return of symptoms and he had noticed that he was losing weight. Four days before admission he had struck his thumb with a hammer and the wound had gone septic. Symptoms had become very pronounced.

PROGRESS: On admission glycosuria was very marked (800gms.) and acetone +++. There was a large sloughing wound in the Rt. hand where an incision had been made. The glands in the axilla were tender. He was



PROGRESS: very emaciated, smelt strongly of acetone, and was  
(Contd.) generally very ill.

Patient was placed on Oatmeal subsequently changed to decreasing diet rich in carbohydrate. In 10 days the acetone was cleared from the urine and the sugar output was much reduced. Meantime the hand improved slowly, but on 20:12:22 another incision was necessary, and again glycosuria was increased, and acetone returned. Thereafter progress was very satisfactory. The hand healed in remarkable fashion and pari passu the sugar output dropped to very small amount. A wonderfully good functional result was obtained with the hand.

READMITTED: 21:3:23. Patient had been very well since previous residence. He had apparently put on weight but this was really due to oedema. He was admitted for insulin treatment.

PROGRESS: Progress was unsatisfactory as the supply of insulin was capricious, and sugar output varied with the dose available. Acidosis gave no trouble. Latterly sufficient insulin was obtained and the urine was quickly rendered sugar-free on an adequate diet. He left hospital on 100:80:120 = 1800, shewing a trace of sugar in the urine.

READMITTED: 27:6:23. With double Chronic Otitis Media and some Bronchitis. He had been working steadily since last dismissal.  
In poor condition: very thin. Deafness very marked.

PROGRESS: On insulin acetone and sugar were rapidly cleared from the urine and progress was uneventful. The discharge from the ears quickly dried up. An attempt was made to drop the insulin and to return to starvation methods, but this was unsuccessful. Later patient required a larger dose of insulin, but he was discovered to be adding to his diet. Toward the end of his residence some experiments were tried with rectal glucose and insulin, but no success was achieved. He left hospital on 80:90:130 = 1850 with 30 units of insulin.



	1920		1921		1922		1923		1923	
	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.
Weight (kilo)	60.	69.5	68.6	57.5	52.6	49.1	59.7	57.3	47.5	50.2
B.S.	-	.20%	$\frac{1}{8}$ 24%	$\frac{1}{8}$ 22%	.20%	.30%	.36%	.23%	.25%	.24%
U.S. (Gms.)	100	Tr.	200	Free	800	77	100	Tr.	245	Trace
U. Acet.	+	-	+	-	++++	+	++	-	++	-

This case illustrates the following points:

- (1) That it is possible for a man to carry on for long periods at full work of an arduous nature on a carefully adjusted equilibrium diet.
- (2) That acidosis and glycosuria are profoundly affected by sepsis, and disappear with the clearing up of the septic condition.
- (3) That the omission of insulin is followed immediately by marked glycosuria. (Chart III).











# DIET

GRAMMES

C. P. F. CALORIES

ONE

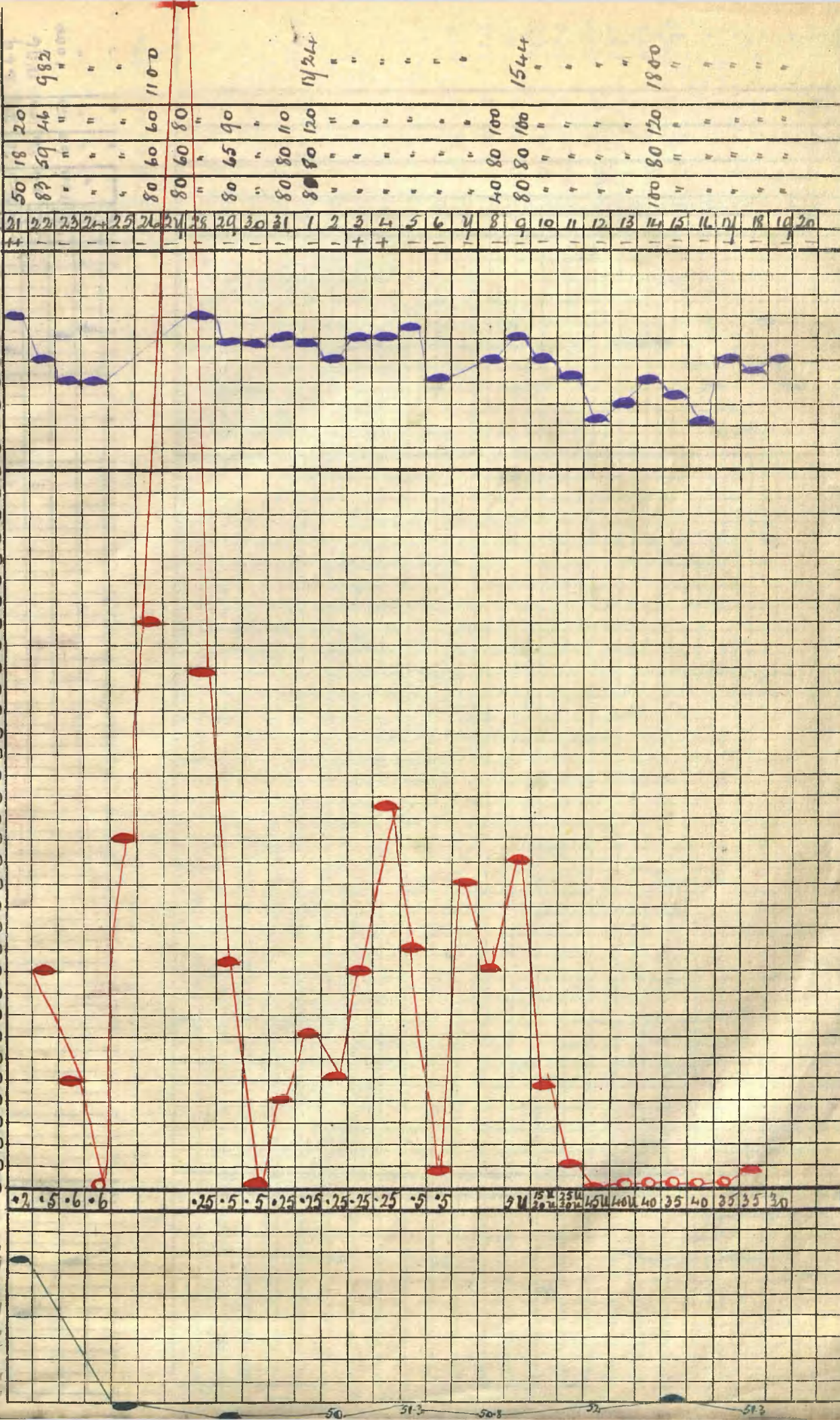
Age.....	0-45
Ward.....	0-40
Journal.....	0-35
Page.....	0-30
BLOOD.....	0-25
SUGAR.....	0-20
PER.....	0-15
CENT.....	0-10
	0-05

TOTAL  
URINARY  
SUGAR

Date of Admission...  
21-3-23

INSULIN UNITS.

WEIGHT  
IN KILOS





Page ..... 0-30 390

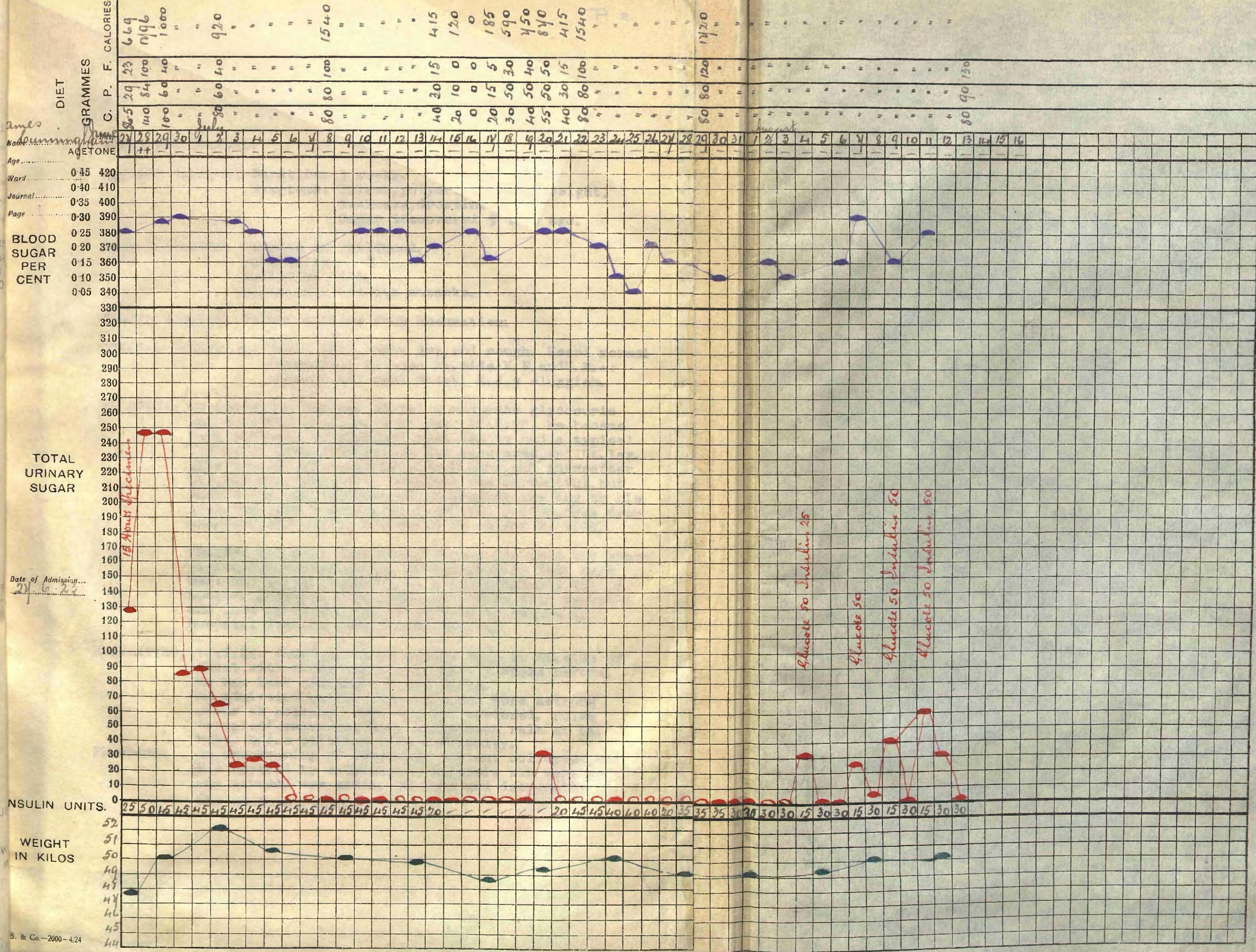
BLOOD	0-25	380
SUGAR	0-20	370
PER	0-15	360
CENT	0-10	350
	0-05	340

TOTAL	230
URINARY	220
SUGAR	210

Date of Admission... 24.6.22

INSULIN UNITS.

WEIGHT IN KILOS	51	50	49
1			
2			
3			
4			
5			
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97			
98			
99			
100			





T.D.

Age 13

School boy.

ADMITTED: 21:2:20.

PRESENT ILLNESS: Duration: 3 weeks.

Symptoms: Thirst, polyuria, loss of weight, weakness, drowsiness.

Sugar discovered 3 days ago.

PAST ILLNESSES: Measles in childhood.

Bronchitis "

Influenza 1919.

Occasional bilious attacks.

FAMILY HISTORY: Father suffers from Rheumatism.

GENERAL CONDITION: Tall: thin. Skin dry and rough. Heart normal  
Lungs-some dulness Rt. side: V. F. and V. R.+:  
rhonchi all over chest. K.J.s sluggish.

PROGRESS: On admission patient shewed a moderate glycosuria and a little acetone. Next day (22:2:20) he became very sick and vomited, and next day he had a typical attack of Tetany affecting the face, arms, and Lt. leg. This passed off completely in  $\frac{1}{2}$  hour, and thereafter the boy was very well. Progress was uneventful, but difficulty was experienced in keeping the boy to his diet. He left hospital on 70:90:160 = 2090, free from symptoms, and having increased in weight by 4.2k.

PROGRESS: Patient was seen frequently during the next two years. Glycosuria was generally present, but he felt very well and worked steadily as a telegraph-boy for 18 months. Polyuria then began to return with weakness and loss of weight, but he refused to return to hospital lest he lose his situation.

READMITTED: 10:2:22. Complaining of severe pain in Rt. side of chest with cough and bloody spit 3 weeks before. He had been confined to bed since.

On admission patient was thin, and bore a strong odour of acetone. Skin dry and powdered. Pulse 'coupled'-extrasystoles. Lungs-much rale at Lt. base. K.J.s elicited with difficulty.

PROGRESS: On the evening of admission patient collapsed, but was revived by stimulants. Acidosis was very marked and oatmeal was given. On this and decreasing diets the acetone rapidly



PROGRESS: cleared from the urine, and glycosuria disappeared.  
(Contd.) Meantime the patient became markedly oedematous: but this disappeared with increasing feeding. It was found impossible to diet him as he persisted in stealing food: but undoubtedly his tolerance had diminished. He went home on 30:60:90 = 1170, free from symptoms.  
There was little doubt that the lung condition was a pneumonia.

READMITTED: 24:9:22. Since dismissal on 23:2:22 patient had been at home, unfit for work. He had not been keeping to his diet. He came back to hospital reluctantly on account of weakness.

Condition: Very emaciated. Skin dry. Smelling of acetone. B.P 70mm.Hg. Dulness and rale Lt. base. K.J.s not elicited.

Progress: Acidosis and glycosuria were again very marked, and again oatmeal was employed. Patient reacted very well, but as before patient would not diet properly, and glycosuria was persistent. He went home on 45:45:65 = 855, with both acetone and sugar in the urine.

READMITTED: 12:3:23. Since dismissal on 23:10:23 patient had dieted strictly from time to time: but he had gone steadily downhill, and latterly he had paid little attention to diet.  
He was admitted terribly emaciated and 'in extremis'. His condition has been described in the 'Insulin' text.

Progress: Insulin treatment was commenced at once with a diet rich in carbohydrate. In three days the acidosis had completely cleared up, and thereafter gave rise to no anxiety. The urinary sugar output was greatly reduced, but an adequate dosage of insulin could not be obtained at that time, and the chart shows very clearly the result of its omission. On 10:4:23 however, the supply became more regular, and progress was thereafter more satisfactory. On 22:4:23 an unsuccessful attempt was made to treat with insulin ointment, an experiment which was repeated twice later ( 22:5:23 and 24:5:23 ). With a fixed diet and dose of insulin ( 30 units ) sugar excretion was little more than a trace, but was breaking diet at this time and the urine could not be kept sugar-free. Later an attempt was made to drop insulin and to treat on a more restricted diet: but tolerance was found to be low and insulin had to be resumed.



Progress: Patient left hospital very well on 70:80:100 = 1500,  
 (Contd.) with a trace of sugar in the urine, taking 20 units  
 of insulin. He had gained 5.5 kilos. in weight.

	1920		1922		1922		1923	
	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.
Weight	29.5k.	33.6k.	29.3k.	29.5k.	28.9k.	27.3k.	24.6k.	29.4k.
Blood								
Sugar	-	-	0.37%	0.21%	0.48%	0.15%	0.45%	0.10%
Urinary								
Sugar	40gms.	Free	138gms.	Free	64gms.	80gms.	252gms.	Trace
Urinary								
Acetone	++	-	++++	-	+++	++	++++	-

A case of severe diabetes, slowly deteriorating under 'Allen' treatment, and going rapidly toward death when diet was broken; and finally being literally saved by insulin administration.







II 0

DIET

GRAMMES

C.	P.	F.	CALORIES
150	150	150	150
200	200	200	200
250	250	250	250
300	300	300	300
350	350	350	350
400	400	400	400
450	450	450	450
500	500	500	500
550	550	550	550
600	600	600	600
650	650	650	650
700	700	700	700
750	750	750	750
800	800	800	800
850	850	850	850
900	900	900	900
950	950	950	950
1000	1000	1000	1000
1050	1050	1050	1050
1100	1100	1100	1100
1150	1150	1150	1150
1200	1200	1200	1200
1250	1250	1250	1250
1300	1300	1300	1300
1350	1350	1350	1350
1400	1400	1400	1400
1450	1450	1450	1450
1500	1500	1500	1500
1550	1550	1550	1550
1600	1600	1600	1600
1650	1650	1650	1650
1700	1700	1700	1700
1750	1750	1750	1750
1800	1800	1800	1800
1850	1850	1850	1850
1900	1900	1900	1900
1950	1950	1950	1950
2000	2000	2000	2000
2050	2050	2050	2050
2100	2100	2100	2100
2150	2150	2150	2150
2200	2200	2200	2200
2250	2250	2250	2250
2300	2300	2300	2300
2350	2350	2350	2350
2400	2400	2400	2400
2450	2450	2450	2450
2500	2500	2500	2500
2550	2550	2550	2550
2600	2600	2600	2600
2650	2650	2650	2650
2700	2700	2700	2700
2750	2750	2750	2750
2800	2800	2800	2800
2850	2850	2850	2850
2900	2900	2900	2900
2950	2950	2950	2950
3000	3000	3000	3000
3050	3050	3050	3050
3100	3100	3100	3100
3150	3150	3150	3150
3200	3200	3200	3200
3250	3250	3250	3250
3300	3300	3300	3300
3350	3350	3350	3350
3400	3400	3400	3400
3450	3450	3450	3450
3500	3500	3500	3500
3550	3550	3550	3550
3600	3600	3600	3600
3650	3650	3650	3650
3700	3700	3700	3700
3750	3750	3750	3750
3800	3800	3800	3800
3850	3850	3850	3850
3900	3900	3900	3900
3950	3950	3950	3950
4000	4000	4000	4000
4050	4050	4050	4050
4100	4100	4100	4100
4150	4150	4150	4150
4200	4200	4200	4200
4250	4250	4250	4250
4300	4300	4300	4300
4350	4350	4350	4350
4400	4400	4400	4400
4450	4450	4450	4450
4500	4500	4500	4500
4550	4550	4550	4550
4600	4600	4600	4600
4650	4650	4650	4650
4700	4700	4700	4700
4750	4750	4750	4750
4800	4800	4800	4800
4850	4850	4850	4850
4900	4900	4900	4900
4950	4950	4950	4950
5000	5000	5000	5000
5050	5050	5050	5050
5100	5100	5100	5100
5150	5150	5150	5150
5200	5200	5200	5200
5250	5250	5250	5250
5300	5300	5300	5300
5350	5350	5350	5350
5400	5400	5400	5400
5450	5450	5450	5450
5500	5500	5500	5500
5550	5550	5550	5550
5600	5600	5600	5600
5650	5650	5650	5650
5700	5700	5700	5700
5750	5750	5750	5750
5800	5800	5800	5800
5850	5850	5850	5850
5900	5900	5900	5900
5950	5950	5950	5950
6000	6000	6000	6000
6050	6050	6050	6050
6100	6100	6100	6100
6150	6150	6150	6150
6200	6200	6200	6200
6250	6250	6250	6250
6300	6300	6300	6300
6350	6350	6350	6350
6400	6400	6400	6400
6450	6450	6450	6450
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6550	6550	6550	6550
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6650	6650	6650	6650
6700	6700	6700	6700
6750	6750	6750	6750
6800	6800	6800	6800
6850	6850	6850	6850
6900	6900	6900	6900
6950	6950	6950	6950
7000	7000	7000	7000
7050	7050	7050	7050
7100	7100	7100	7100
7150	7150	7150	7150
7200	7200	7200	7200
7250	7250	7250	7250
7300	7300	7300	7300
7350	7350	7350	7350
7400	7400	7400	7400
7450	7450	7450	7450
7500	7500	7500	7500
7550	7550	7550	7550
7600	7600	7600	7600
7650	7650	7650	7650
7700	7700	7700	7700
7750	7750	7750	7750
7800	7800	7800	7800
7850	7850	7850	7850
7900	7900	7900	7900
7950	7950	7950	7950
8000	8000	8000	8000
8050	8050	8050	8050
8100	8100	8100	8100
8150	8150	8150	8150
8200	8200	8200	8200
8250	8250	8250	8250
8300	8300	8300	8300
8350	8350	8350	8350
8400	8400	8400	8400
8450	8450	8450	8450
8500	8500	8500	8500
8550	8550	8550	8550
8600	8600	8600	8600
8650	8650	8650	8650
8700	8700	8700	8700
8750	8750	8750	8750
8800	8800	8800	8800
8850	8850	8850	8850
8900	8900	8900	8900
8950	8950	8950	8950
9000	9000	9000	9000
9050	9050	9050	9050
9100	9100	9100	9100
9150	9150	9150	9150
9200	9200	9200	9200
9250	9250	9250	9250
9300	9300	9300	9300
9350	9350	9350	9350
9400	9400	9400	9400
9450	9450	9450	9450
9500	9500	9500	9500
9550	9550	9550	9550
9600	9600	9600	9600
9650	9650	9650	9650
9700	9700	9700	9700
9750	9750	9750	9750
9800	9800	9800	9800
9850	9850	9850	9850
9900	9900	9900	9900
9950	9950	9950	9950
10000	10000	10000	10000

Name T.D.

Age 0.45

Ward 420

Journal 0.40

Page 0.35

BLOOD SUGAR PER CENT

0.30

0.25

0.20

0.15

0.10

0.05

0.00

0.00

0.00

0.00

0.00

0.00

0.00

0.00

0.00

0.00

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0.00

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ACETONE

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50

51

52

53

54



# DIET

Name: TD Date: 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23

ACETONE

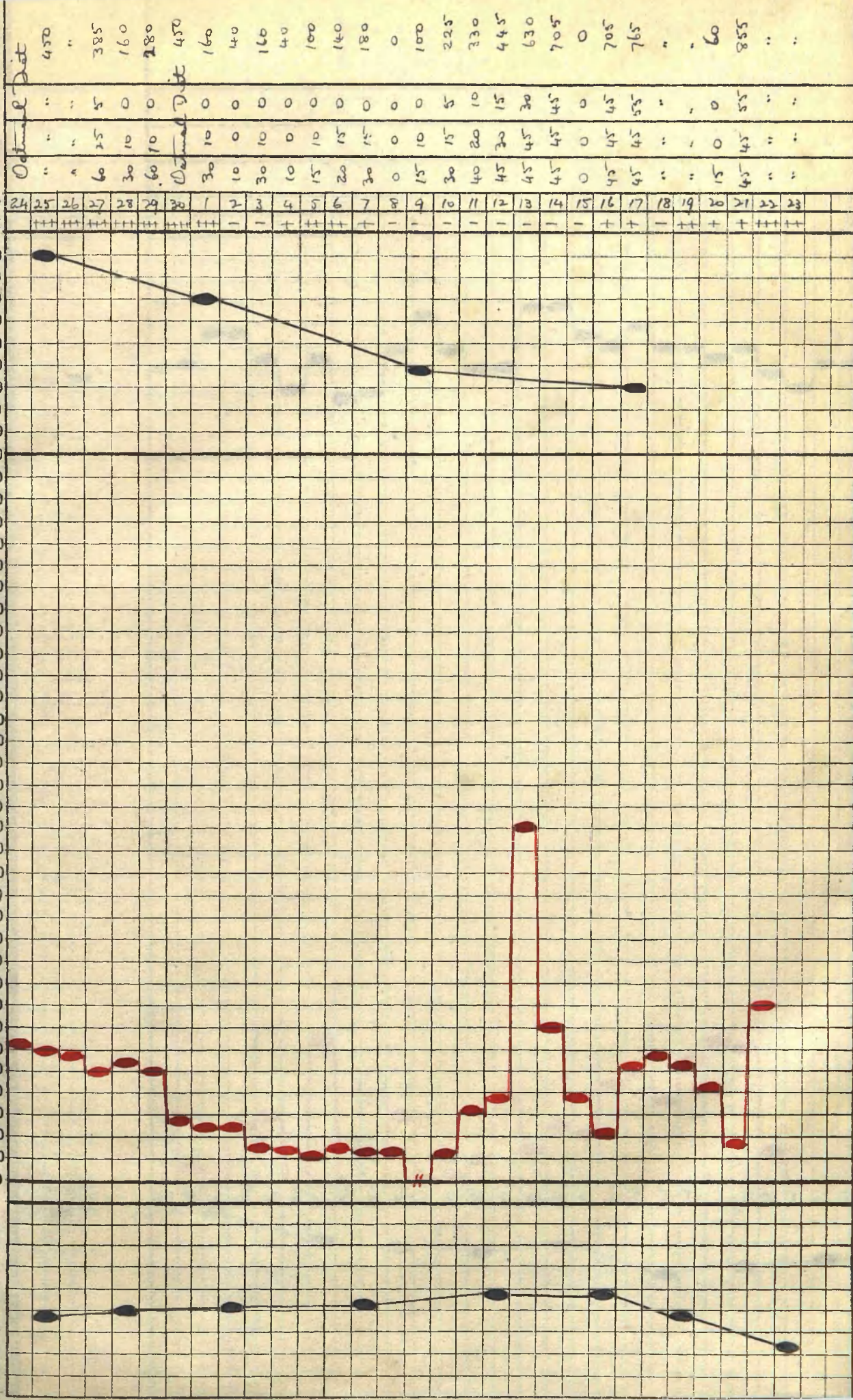
Age: 0.45 420  
Ward: 0.40 410  
Journal: 0.35 400  
Page: 0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

330  
320  
310  
300  
290  
280  
270  
260  
250  
240  
230  
220  
210  
200  
190  
180  
170  
160  
150  
140  
130  
120  
110  
100  
90  
80  
70  
60  
50  
40  
30  
20  
10  
0

INSULIN UNITS.

WEIGHT  
IN KILOS

33  
32  
31  
30  
29  
28  
27  
26  
25





DIET  
GRAMMES  
C. P. F. CALORIES

Name TD Date 12.3.23

Age 42  
Ward 0.45 420  
Journal 0.40 410  
Page 0.35 400  
0.30 390  
0.25 380  
BLOOD SUGAR PER CENT  
0.20 370  
0.15 360  
0.10 350  
0.05 340

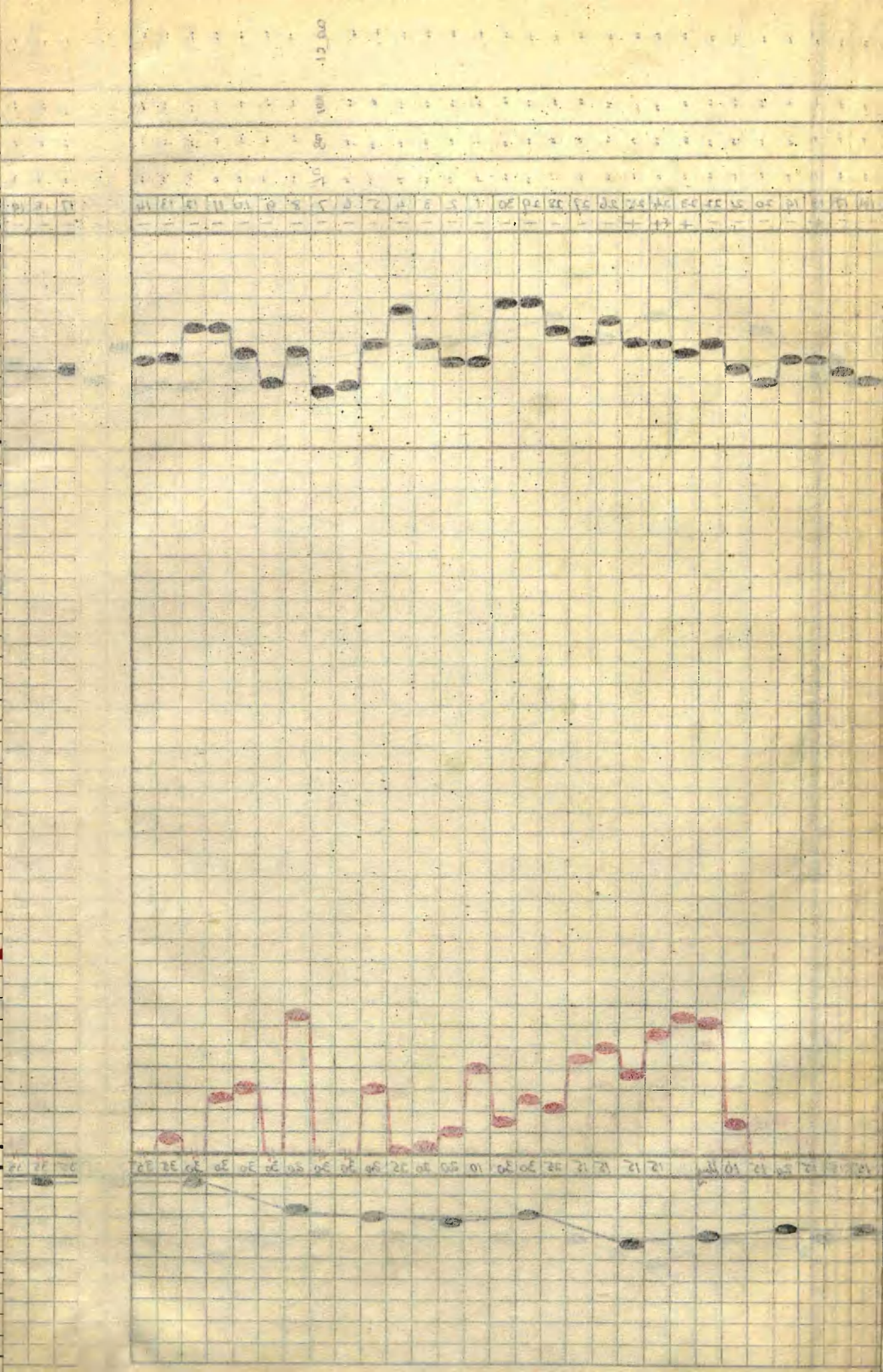
330  
320  
310  
300  
290  
280  
270  
260  
250  
240  
230  
TOTAL URINARY SUGAR  
220  
210  
200  
190  
180  
170  
160  
150

Date of Admission 12.3.23

140  
130  
120  
110  
100  
90  
80  
70  
60  
50  
40  
30  
20  
10  
0

INSULIN UNITS.

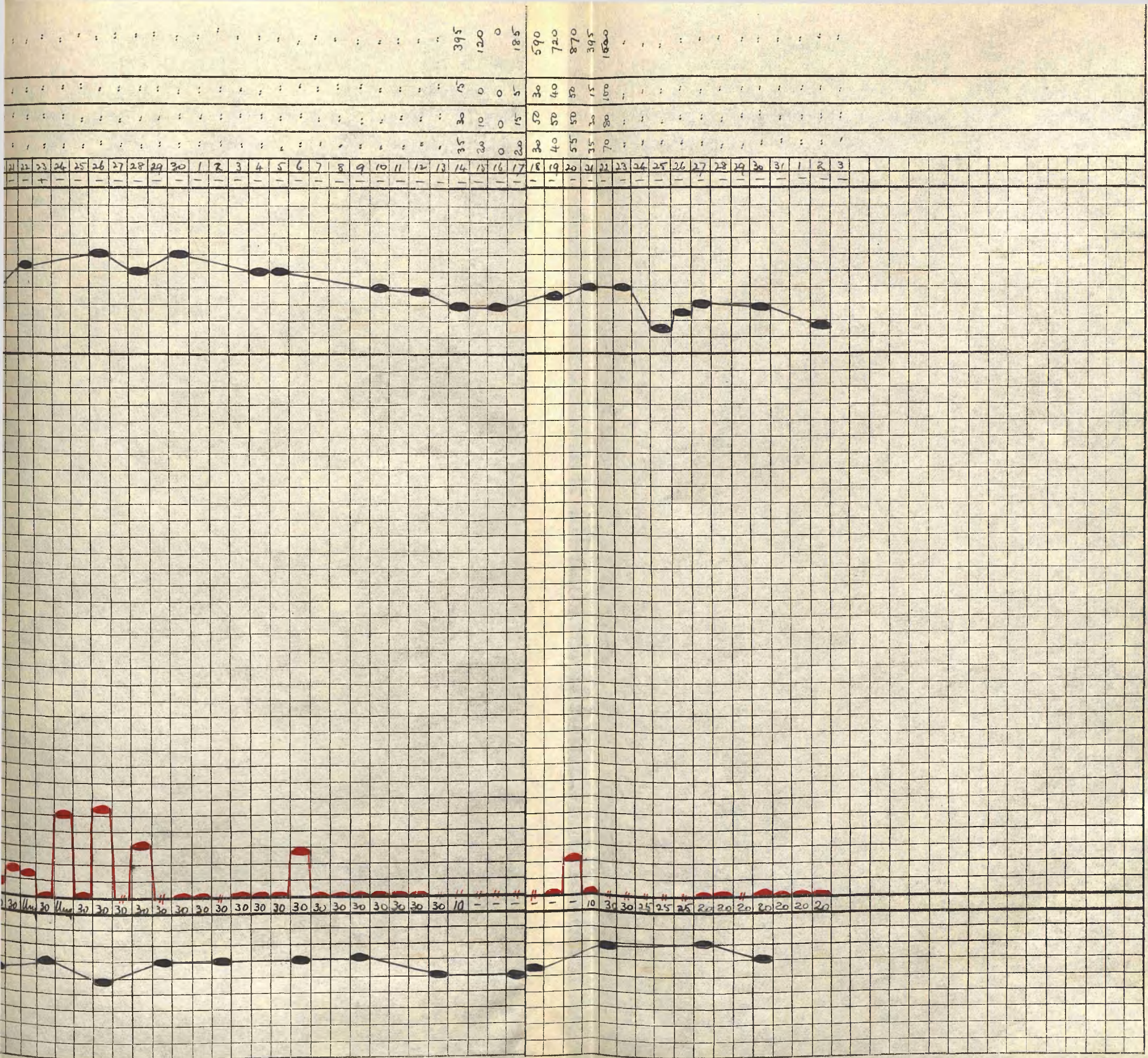
WEIGHT IN KILOS  
29  
28  
27  
26  
25  
24  
23  
22  
21





[illegible][illegible][illegible]







W. McN.

Age 40

Clerk.

ADMITTED: 8:3:21.

PRESENT ILLNESS: Duration: 3 years.

Symptoms: Thirst, polyuria, weakness, loss of weight, sleeplessness.

Sugar discovered Dec. 1918: dieted with considerable improvement, but latterly symptoms had returned.

PAST ILLNESSES: Scarlet Fever in childhood.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Tall: thin: sallow: looking ill. Skin dry, but no skin lesions. Heart normal. Lungs - impaired percussion note Rt. apex: R.M. deficient. K.J.s not elicited. Eyes normal.

PROGRESS: Patient was given a day on ordinary diet to determine the extent of the glycosuria, and this was followed by decreasing diet to starvation. The sugar was rapidly cleared from the urine. Acidosis gave rise to no anxiety. With increasing feeding glycosuria remained absent save on two occasions. Patient went home on 109:99:104 = 1858, free from symptoms.

READMITTED: 26:5:23. Since dismissal on 2:4:21 patient had dieted himself with the utmost strictness. He had collected together in book form the records of the food values of every meal taken during that time, the type of food and the amounts being given in detail. He had tended to err on the side of undernutrition throughout, but he had avoided starve days as being too weakening. If glycosuria returned at any time he found that he could banish it by very vigorous exercise. He had been regularly at work throughout.

GENERAL CONDITION: Very thin. Looking tired and ill. Skin very dry. Heart normal. Lungs - no dulness to percussion: rale over chest, + at Rt. apex: considerable cough, no sputum. K.J.s present but elicited with difficulty. Urine - albumen ++.



**PROGRESS:** As patient's condition was far from satisfactory insulin was commenced on admission. On a diet fairly rich in carbohydrate the insulin was rapidly increased, but even with 65 units and a reduced carbohydrate intake the urine could not be rendered sugar-free. Subsequently the dose was reduced to 55 units, and with a diet around 68:92:132 = 1825 the glycosuria was kept within reasonable limits ( Trace - 30gms.) Patient went home much improved in every way. The albuminuria cleared up on the 10th. day and did not recur.

	1921		1923	
	Ad.	Dis.	Ad.	Dis.
Weight	54k.	54k.	56.6k.	
Blood Sugar	0.40%	0.18%	0.25%	0.18%
Urinary Sugar	280gms.	Free	190gms.	20gms.
Urinary Acetone	+	-	-	-

Seen subsequently the urine was sugar-free on an unchanged diet but with an insulin dose of 40 units. The weight had increased and patient felt very well. The lung condition apparently remained quiescent.

- A case showing
- (1) a satisfactory result on diet alone,
  - (2) a loss of tolerance and marked weakness following prolonged under-feeding,
  - (3) a satisfactory result, ultimately, with insulin.

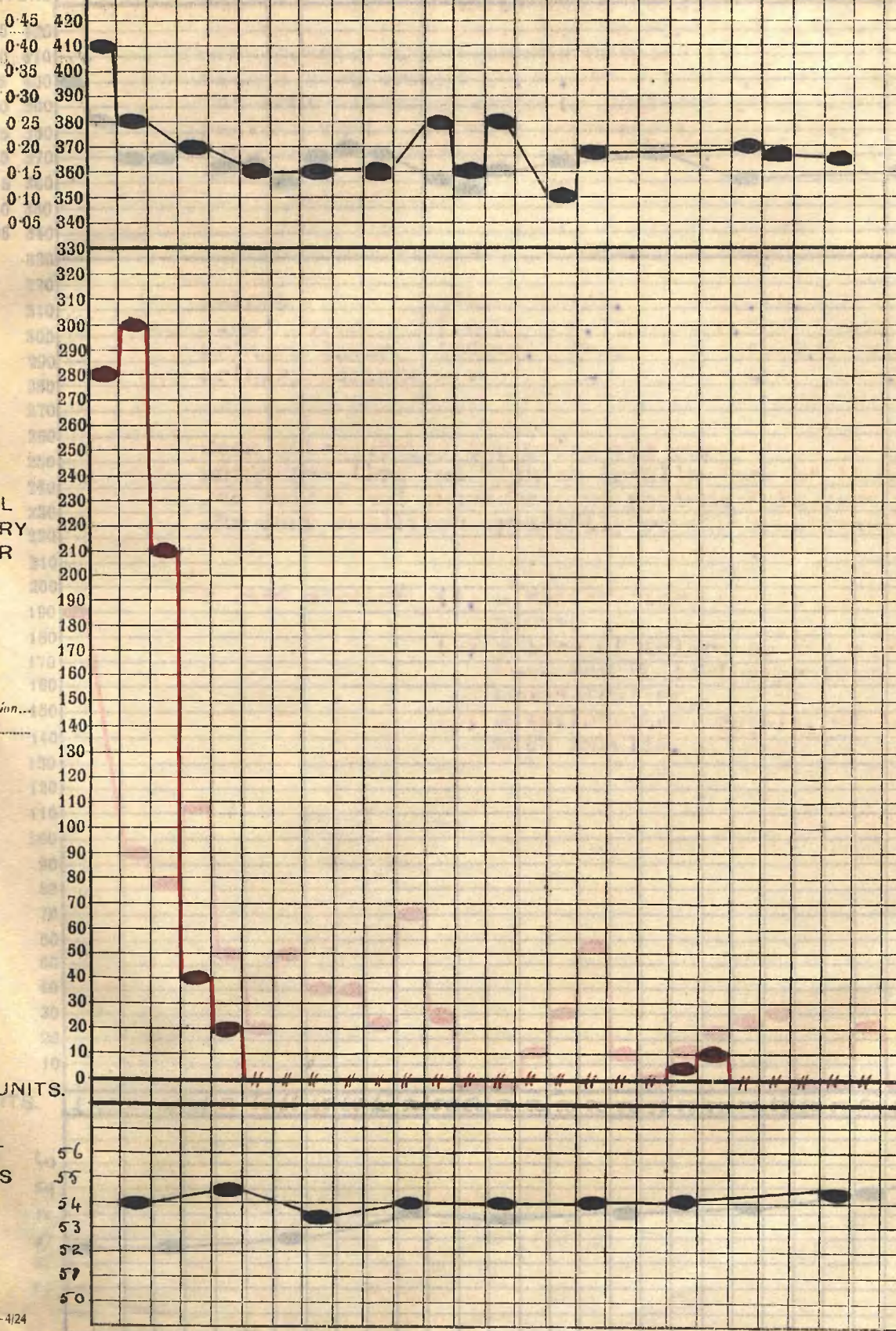
Name M. N. Date 8/9/21  
 Age 45  
 Ward 420  
 Journal 410  
 Page 400  
 BLOOD SUGAR PER CENT  
 0.45 420  
 0.40 410  
 0.35 400  
 0.30 390  
 0.25 380  
 0.20 370  
 0.15 360  
 0.10 350  
 0.05 340  
 0.00 330  
 0.00 320  
 0.00 310  
 0.00 300  
 0.00 290  
 0.00 280  
 0.00 270  
 0.00 260  
 0.00 250  
 0.00 240  
 0.00 230  
 0.00 220  
 0.00 210  
 0.00 200  
 0.00 190  
 0.00 180  
 0.00 170  
 0.00 160  
 0.00 150  
 0.00 140  
 0.00 130  
 0.00 120  
 0.00 110  
 0.00 100  
 0.00 90  
 0.00 80  
 0.00 70  
 0.00 60  
 0.00 50  
 0.00 40  
 0.00 30  
 0.00 20  
 0.00 10  
 0.00 0  
 Date of Admission 8/3/21

INSULIN UNITS.

WEIGHT IN KILOS

DIET	GRAMMES	C.	P.	F.	CALORIES
		100	100	100	2900
		186	69	55	1515
		126	34	12	748
		36	12	2	210
		30	0	0	120
		0	0	0	0
		0	0	0	0
		10	0	0	40
		20	5	5	145
		30	15	5	225
		40	20	5	285
		53	24	12	390
		71	36	18	
		81	44	23	
		92	42	30	
		109	60	32	
		0	0	0	
		119	63	33	
		134	76	44	
		145	76	44	
		160	70	48	
		40	20	5	285
		92	80	62	1246
		99	68	100	1568
		99	86	113	1757
		109	99	114	1858

ACETONE





GRAMMES

**CALORIES**

1542 1574 1196 1302 1302 1490 1591 1624 1624 1768 " 1757 1808 1752 1688 " 1669 1708 1740 1772 1704 1708 1792 1827 1740 1872 1808 1828 1828 1828 "

[illegible]

26	27	28	29	30	31	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
----	----	----	----	----	----	---	---	---	---	---	---	---	---	---	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----

[illegible]

The graph displays a series of data points connected by a line, plotted on a grid. The data starts at a high value on the left, drops sharply to a lower level, and then continues with several smaller fluctuations before ending at a point slightly higher than the previous one.

The graph displays a series of data points connected by a red line on a grid. The vertical axis (y-axis) ranges from 0 to 10, and the horizontal axis (x-axis) ranges from 0 to 10. The data points are approximately as follows:

X-axis	Y-axis
0.5	9.5
1.5	6.5
2.5	6.0
3.5	7.5
4.5	6.5
5.5	5.5
6.5	6.5
7.5	6.5
8.5	5.5
9.5	7.0
10.5	6.0
11.5	5.0
12.5	6.0
13.5	7.0
14.5	5.0
15.5	5.0
16.5	5.5
17.5	5.5
18.5	6.0
19.5	5.0
20.5	5.0
21.5	5.5
22.5	6.0
23.5	5.0
24.5	5.0
25.5	5.5
26.5	5.5
27.5	6.0
28.5	5.0
29.5	5.0
30.5	5.5
31.5	5.5
32.5	6.0
33.5	5.0
34.5	5.0
35.5	5.5
36.5	5.5
37.5	6.0
38.5	5.0
39.5	5.0
40.5	5.5
41.5	5.5
42.5	6.0
43.5	5.0
44.5	5.0
45.5	5.5
46.5	5.5
47.5	6.0
48.5	5.0
49.5	5.0
50.5	5.5
51.5	5.5
52.5	6.0
53.5	5.0
54.5	5.0
55.5	5.5
56.5	5.5
57.5	6.0
58.5	5.0
59.5	5.0
60.5	5.5
61.5	5.5
62.5	6.0
63.5	5.0
64.5	5.0
65.5	5.5
66.5	5.5
67.5	6.0
68.5	5.0
69.5	5.0
70.5	5.5
71.5	5.5
72.5	6.0
73.5	5.0
74.5	5.0
75.5	5.5
76.5	5.5
77.5	6.0
78.5	5.0
79.5	5.0
80.5	5.5
81.5	5.5
82.5	6.0
83.5	5.0
84.5	5.0
85.5	5.5
86.5	5.5
87.5	6.0
88.5	5.0
89.5	5.0
90.5	5.5
91.5	5.5
92.5	6.0
93.5	5.0
94.5	5.0
95.5	5.5
96.5	5.5
97.5	6.0
98.5	5.0
99.5	5.0
100.5	5.5
101.5	5.5
102.5	6.0
103.5	5.0
104.5	5.0
105.5	5.5
106.5	5.5
107.5	6.0
108.5	5.0
109.5	5.0
110.5	5.5
111.5	5.5
112.5	6.0
113.5	5.0
114.5	5.0
115.5	5.5
116.5	5.5
117.5	6.0
118.5	5.0
119.5	5.0
120.5	5.5
121.5	5.5
122.5	6.0
123.5	5.0
124.5	5.0
125.5	5.5
126.5	5.5
127.5	6.0
128.5	5.0
129.5	5.0
130.5	5.5
131.5	5.5
132.5	6.0
133.5	5.0
134.5	5.0
135.5	5.5
136.5	5.5
137.5	6.0
138.5	5.0
139.5	5.0
140.5	5.5
141.5	5.5
142.5	6.0
143.5	5.0
144.5	5.0
145.5	5.5
146.5	5.5
147.5	6.0
148.5	5.0
149.5	5.0
150.5	5.5
151.5	5.5
152.5	6.0
153.5	5.0
154.5	5.0
155.5	5.5
156.5	5.5
157.5	6.0
158.5	5.0
159.5	5.0
160.5	5.5
161.5	5.5
162.5	6.0
163.5	5.0
164.5	5.0
165.5	5.5
166.5	5.5
167.5	6.0
168.5	5.0
169.5	5.0
170.5	5.5
171.5	5.5
172.5	6.0
173.5	5.0
174.5	5.0
175.5	5.

[illegible]

Year	Number of people (millions)
1950	10
1955	10
1960	11
1965	14
1970	13
1975	14
1980	15
1985	18
1990	22



J.C.

Age 17.

Apprentice Cartwright.

ADMITTED: 13:3:21.

PRESENT ILLNESS: Duration: 5 weeks.

Symptoms: Thirst, hunger, polyuria, loss of weight, weakness, dimness of vision.

PAST ILLNESSES: Diphtheria in childhood.

Measles in childhood.

"Cerebral Attack" 1920: unconscious: ? origin.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Physique fair: thin. Site of old haemorrhage into  
Right retina marked by large pigmented area.  
Physical examination otherwise negative.

PROGRESS: Progress was very satisfactory at first, sugar and acetone  
being quickly cleared from the urine on a low dietary.  
It was found impossible, however, to find the Carbohydrate  
tolerance or to arrive at an equilibrium diet as patient  
persistently stole food. He left hospital free from  
symptoms and with weight unchanged on 30:75:880 = 1410.

READMITTED: 26:5:21. Since dismissal on 30:4:21 he had been unfit to  
return to work. His previous symptoms had returned, and  
in addition he had experienced cramp-like pains in the  
feet when walking.  
The physical condition was unchanged. Wass. React. negative.

PROGRESS: Again glycosuria and other symptoms disappeared under  
suitable diet. On this occasion he ate unauthorised food  
on two occasions only, and he was able to leave hospital  
on 90:80:110 = 1670, without sugar or acetone in the urine.

READMITTED: 21:4:23. Since discharge on 20:6:21 he had been working  
steadily. He complained of slight polyuria and weakness.  
He appeared thinner, and although his weight was up he  
had grown 5cms. in the interval.  
Physical examination revealed nothing further save that  
the knee jerks could not now be elicited.

PROGRESS: As there was evidence of considerable acidosis and glycos-  
uria insulin was commenced on the 5th. day. On 25 units  
the urine was sugar and acetone-free. Some difficulty  
was experienced at this time in obtaining insulin, and on  
2:5:23 an insulin ointment preparation was tried with  
apparently satisfactory results. With new supplies inject-  
ions were resumed; but patient tired of the treatment and



PROGRESS: left hospital irregularly on C 70: P 60: F 80 = 1240 with  
(Contd.) 25 units of insulin.

	1921		1921		1923	
	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.
Weight	43.2k.	43.2k.	42.5k.	40.8k.	42.5k.	42.5k.
Blood Sugar	0.24%	0.18%	0.20%	0.24%	0.20%	0.08%
Urinary Sugar	112gms.	Free	124gms.	Free	32gms.	Free
Urinary Acetone	+	-	-	-	++	-

A case of moderately severe diabetes which responded very well to Allen treatment, and where, in spite of failure to diet strictly, the patient was able to carry on at work for almost 2 years. His response to insulin, too, was excellent, and had he been content to continue the treatment the prognosis would have been good. As it was the outlook was unfavourable.

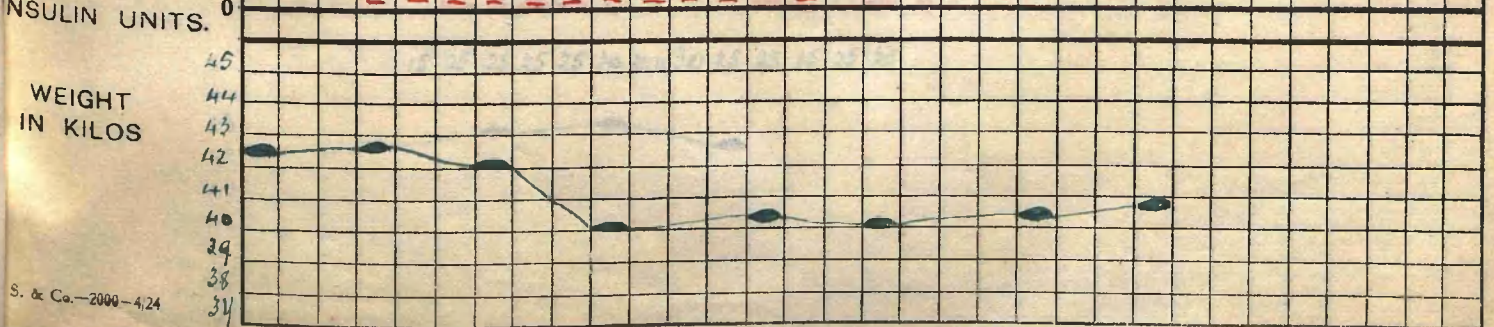
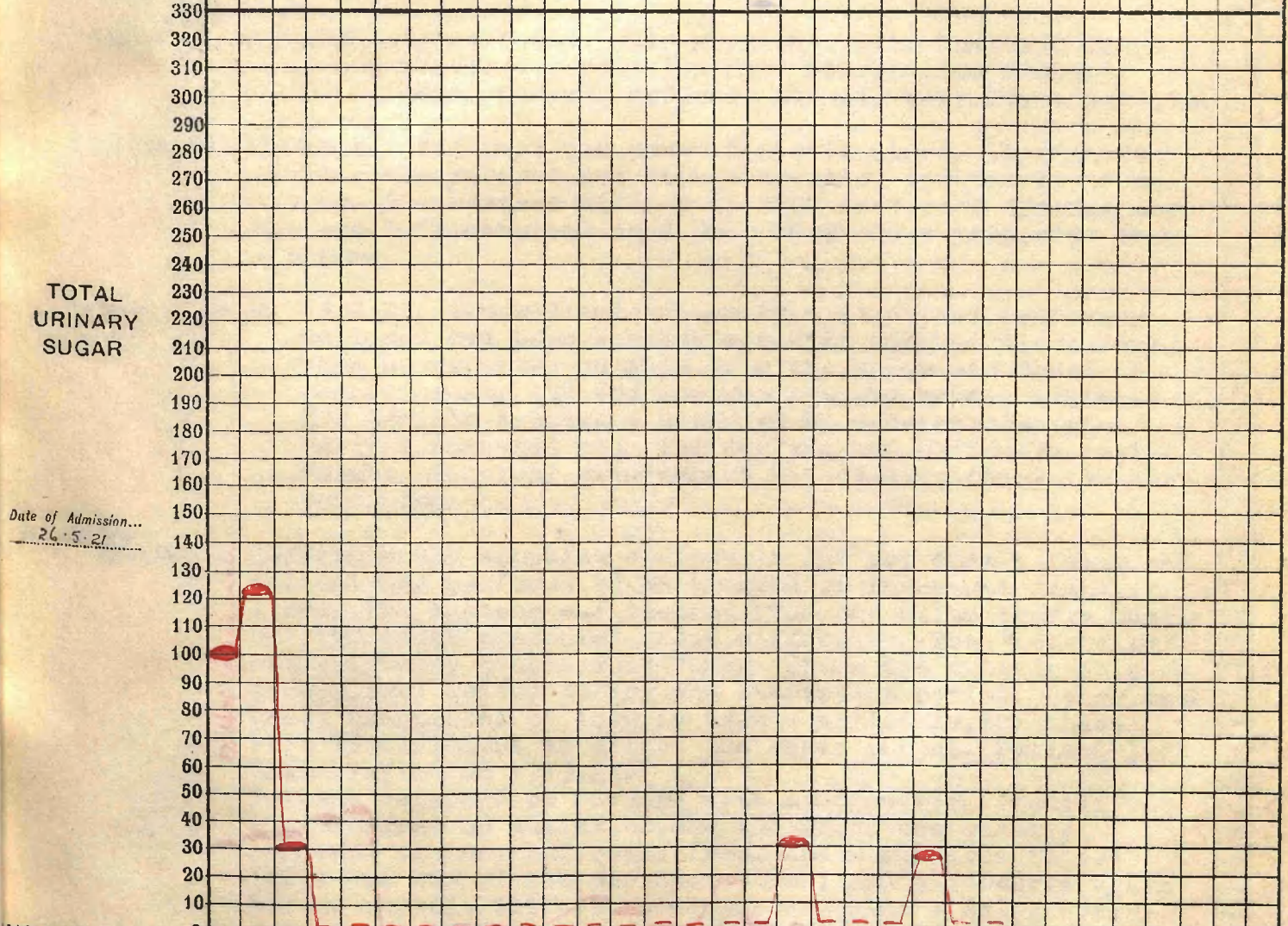
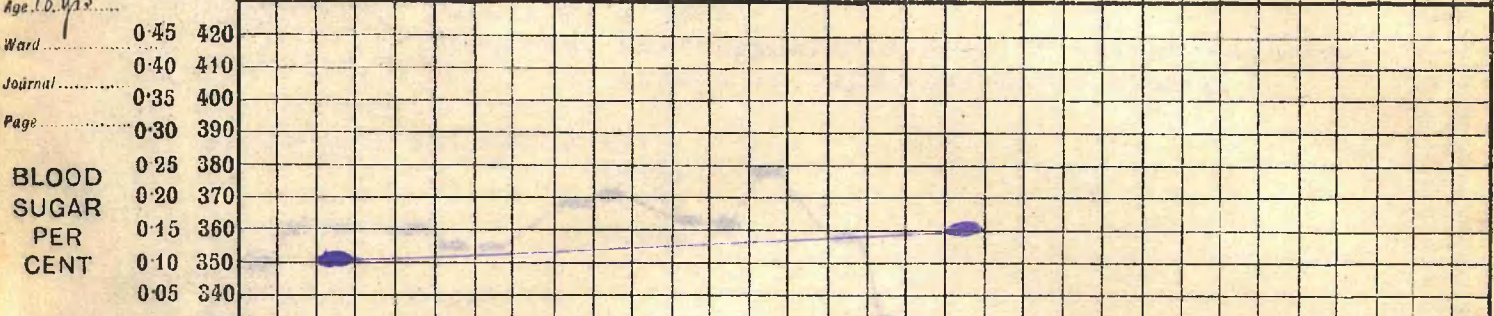






DIET		GRAMMES		F. CALORIES	
		C.	P.	P.	
		100	106	110	1814
		60	25	5	385
		30	10	0	120
		10	0	0	40
		0	0	0	0
		10	0	0	40
		20	0	0	80
		30	0	5	120
		40	15	0	220
		50	20	5	
		60	20	10	335
		70	30	15	405
		80	40	20	0
		90	50	30	830
		100	60	35	955
		100	70	45	1085
		110	70	55	1215
		0	0	0	0
		90	70	40	0
		90	70	85	"
		90	70	100	"
		0	0	0	0
		"	"	"	"
		90	80	110	

Name Gray Date 26 ACETONE -  
 Age 18 yrs 27 -  
28 -  
29 -  
30 -  
31 -  
1 -  
2 -  
3 -  
4 -  
5 +  
6 -  
7 -  
8 -  
9 -  
10 -  
11 -  
12 -  
13 -  
14 -  
15 -  
16 -  
17 -  
18 -  
19 -  
20 +









W.P.

Age 34

Clerk.

ADMITTED: 6:4:21. severe pain in the abdomen. His regular 5 unit

PRESENT ILLNESS: Duration: 1 year.

Symptoms: Thirst, polyuria, loss of weight, weakness, cramps in legs, dimness of vision.

PAST ILLNESSES: Influenza 1911. normally garrulous. Skin very dry.

" " 1912. small. The abdomen contained some

" " 1913. long abnormal could be palpated.

" " 1919. There was a ... Urine - turbid, containing pus in amount and some blood; no casts. Sugar 5%.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Spare build: fair physique. Skin healthy. Lungs - impaired percussion note Rt. apex, few rhonchi. Heart normal. Reflexes normal. Wass. React. negative

PROGRESS: Treatment followed the usual dietetic lines. Glycosuria rapidly disappeared, but with starvation acetonuria became marked. The acetone cleared up with increased feeding, and patient left hospital on C 50:P 80:F 100 = 1420, free from symptoms.

READMISSION: 15:2:23. Since dismissal on 30:4:21 he had been very well, and had been working steadily without discomfort. When he reported on this date the urine was found to contain sugar 15%, and acetone +++, and he was admitted for insulin treatment although he made no complaint. Patient appeared much thinner and had obviously lost weight. Physical examination revealed no change in his condition.

PROGRESS: Unfortunately supplies of insulin did not come to hand and patient had perforce to be treated on the usual dietetic lines. The acetone was cleared from the urine fairly easily but the sugar presented great difficulty. The "tolerance" had evidently become much lower. There was considerable generalised oedema during the starvation period. Later small quantities of insulin became available, but these were insufficient to affect the sugar output. Patient left dissatisfied on 17:3:23.

Patient returned on 28:3:23 with acetone +++. Insulin was administered on admission, and the urine was quickly rendered acetone and sugar-free. Again supplies failed (this was one of the earliest cases) and glycosuria again became marked: but with adequate supplies a satisfactory result was soon attained. He left hospital on C60: P80:

F120 = 1640,

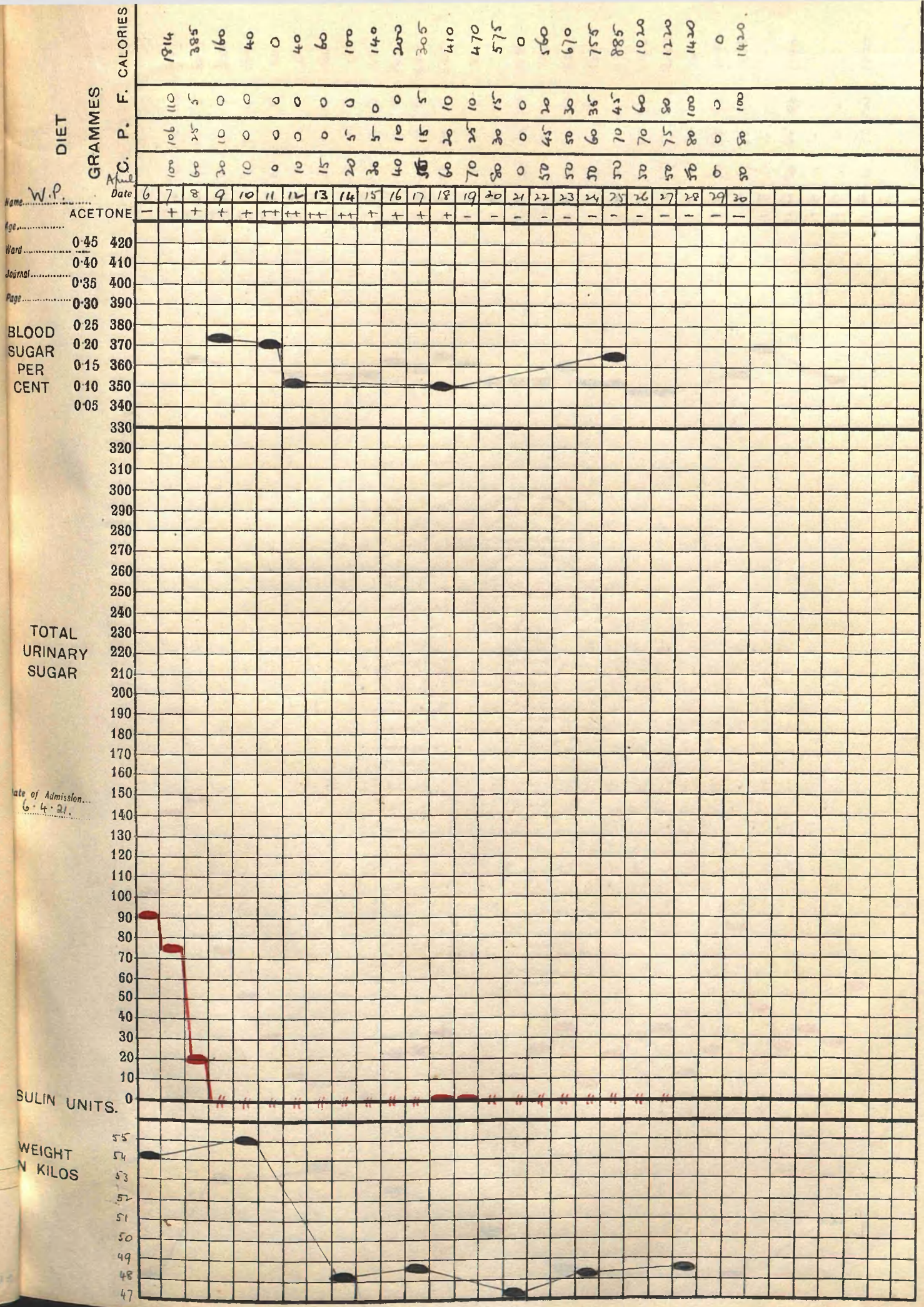


PROGRESS: with 5 units of insulin, free from symptoms.  
(Contd.)

READMITTED: 16:4:24. Well and at work since discharge on 16:4:23 until 2 weeks before when he was compelled to lie up with severe pains in the abdomen. His regular 5 unit dose of insulin was stopped and this was followed at once by a return of intense thirst, dryness in the mouth, together with some mental confusion. Patient looked pale and ill. Greatly emaciated: some oedema of the feet. Quite intelligent, but speech slow and halting though normally garrulous. Skin very dry. Lungs normal. Pulse small. The abdomen contained some free fluid, but nothing abnormal could be palpated. There was a slight balanitis. Urine - turbid, containing pus in amount and some blood: no casts. Sugar 5%.

PROGRESS: Patient was put on C60 :P50 :F50 with 10 units of insulin. He improved very rapidly, and next day was able to sit up and talk in his usual manner. The skin became moist, and the balanitis cleared up. Pus persisted in the urine in amount. The behaviour of the blood-sugar and urinary sugar was very curious, and in consequence the dose of insulin varied considerably - 5 to 25 units. On 26:4:24 a tumour was felt in the abdomen in the region of the right kidney. Oedema and ascites were soon so marked that it could not be felt. Meantime the urine contained pus ++, but no T.B. With increasing anasarca diet and insulin dosage were increased. On 15:5:24 the patient developed an abscess on the back, over the right kidney. This was opened on 19:5:24, and found to be perinephric. Following operation the general condition improved somewhat, although pus persisted in the urine. On 6:6:24 the oedema had completely disappeared, and there was only a trace of sugar in the urine. Healing was slow but satisfactory. The diabetic progress, however, was not good after 13:6:24, and the pyuria became even more marked. On 27:6:24 patient elected to go home. The glycosuria was much worse<sup>on</sup> an unchanged dose of insulin ( 35 units ) with a reduced diet - 60:70:130 = 1790. Although the renal sepsis was active the general condition contraindicated surgical interference.

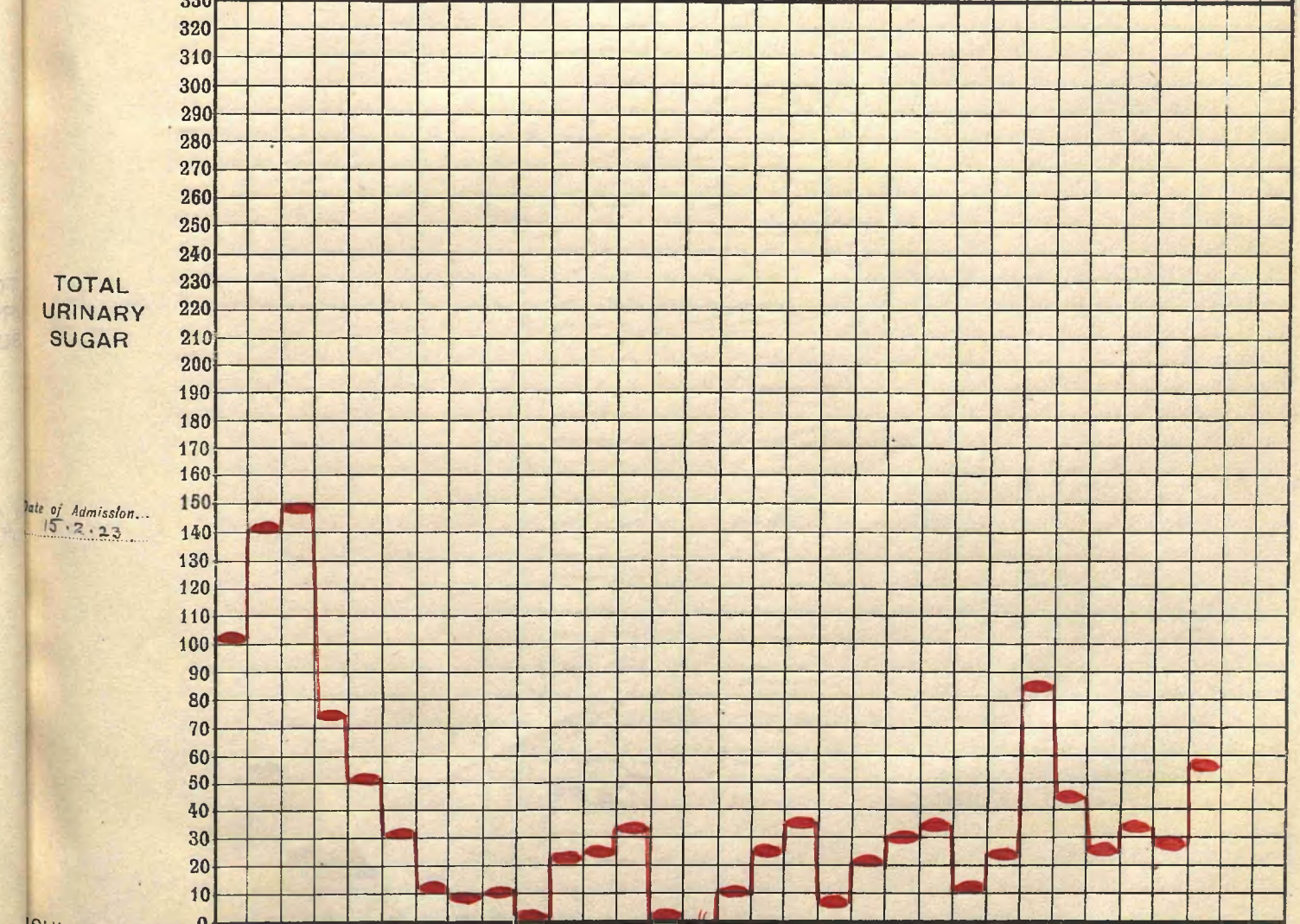
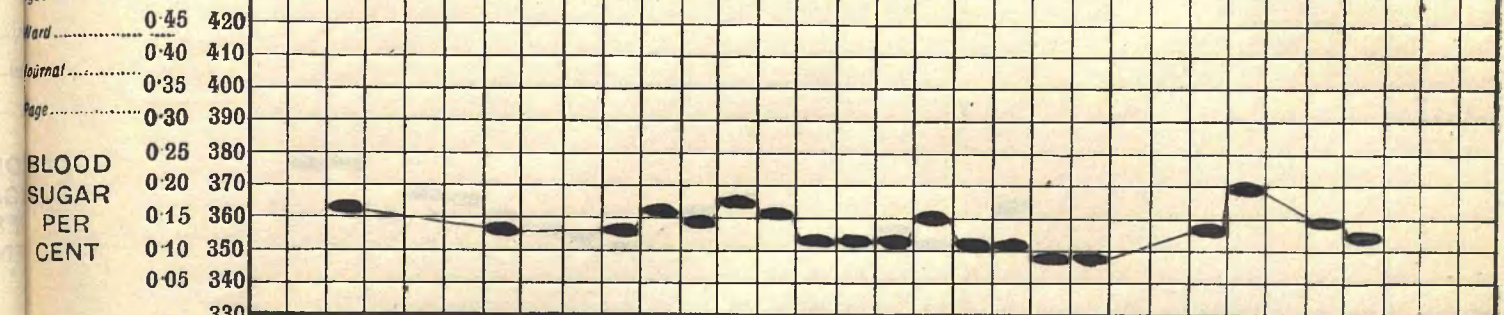




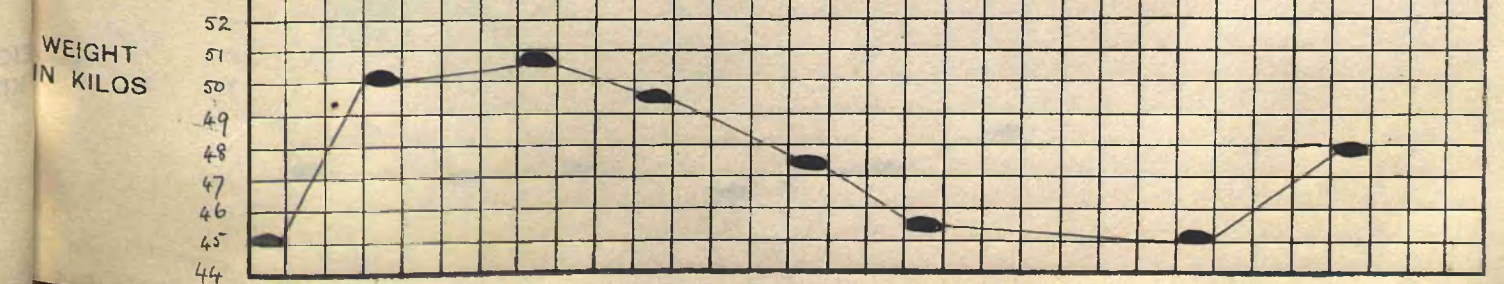


DIET	GRAMMES	P. F. CALORIES
W.P.	100	1780
	25	385
	10	280
	10	160
	0	40
	0	0
	10	160
	0	0
	0	40
	10	160
	15	220
	20	325
	0	0
	20	465
	40	750
	50	920
	60	1100
	60	1100
	65	1190
	65	1456
	65	1190
	65	1456
	65	1190
	65	1190
	70	1320

Date	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
------	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	----	---	---	---	---	---	---	---	---	---	----	----	----	----	----	----	----	----



INSULIN UNITS.



Date of Admission... 15.2.23











V.G.

Age 31

Printer.

ADMITTED: 17:3:22.

PRESENT ILLNESS: Duration: 6 months.

Symptoms: Thirst, polyuria, weakness, dimness of vision, constipation.

PREVIOUS ILLNESSES: Measles in childhood.

Bronchitis 1903.

G.S.W. Left arm: elbow excised: septic 1917 and 1921.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Fairly well developed: left arm atrophied. Skin normal. Tongue coated. Reflexes normal.

PROGRESS: Patient was treated on the usual dietetic lines. Response was excellent, acetone and sugar rapidly disappearing from the urine, and remaining absent. He left hospital free from symptoms on C 85:P 110:F 150 = 2120.

READMITTED: 21:4:23. He had been very well since discharge on 14:4:22 and had worked without a break since that date. He was asked to return for insulin treatment if deemed necessary.

PROGRESS: As sugar was present in the urine in amount, and acidosis was marked insulin was commenced on the 4th. day, and increased rapidly to 30 units. The acetonuria rapidly cleared up, but the glycosuria was more obstinate, due partly to the interpolation of 2 days insulin ointment treatment. On 25 units, however, the urine was constantly sugar-free, and thereafter the dose of insulin was reduced until no insulin was being given without the return of glycosuria. The prolonged administration of insulin had apparently raised his sugar tolerance very markedly. He was dismissed on 80:80:110 = 1630 without insulin, free from symptoms.

READMITTED: 3:10:23. Since discharge on 29:6:23 patient had been very well till 4 days before admission when he developed a carbuncle. All his old symptoms returned. He was very thin and ill: skin dry: tongue coated: odour of acetone in the breath. There was a large carbuncle over the left shoulder, recently incised.

Progress: Acetone and sugar were present in large amount in the urine, and insulin was commenced at once.



Progress: The dose was increased rapidly to 50 units, at which point (Contd.) all symptoms disappeared. The healing of the carbuncle proceeded rapidly and without incident. He left hospital on 80:100:140 = 2040, but now he required 35 units of insulin. The general condition was much improved.

	1922		1923 QOWE		Oct. 1923	
	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.
Weight	53.5k.	52.4k.	51.5k.	51.4k.	40.2k.	50.9k.
Blood Sugar	0.24%	0.20%	0.36%	0.25%	0.28%	0.17%
Urinary Sugar	190gms.	Free	85gms.	Trace	220gms.	Free
Urinary Acetone	++	-	+++	-	++++	-

This case illustrates clearly the following:

- (1) A very satisfactory result with Allen treatment.
- (2) The apparent raising of the sugar tolerance by the prolonged administration of insulin.
- (3) The effect of sepsis in upsetting metabolism, causing a marked reduction in sugar tolerance and profound acidosis.



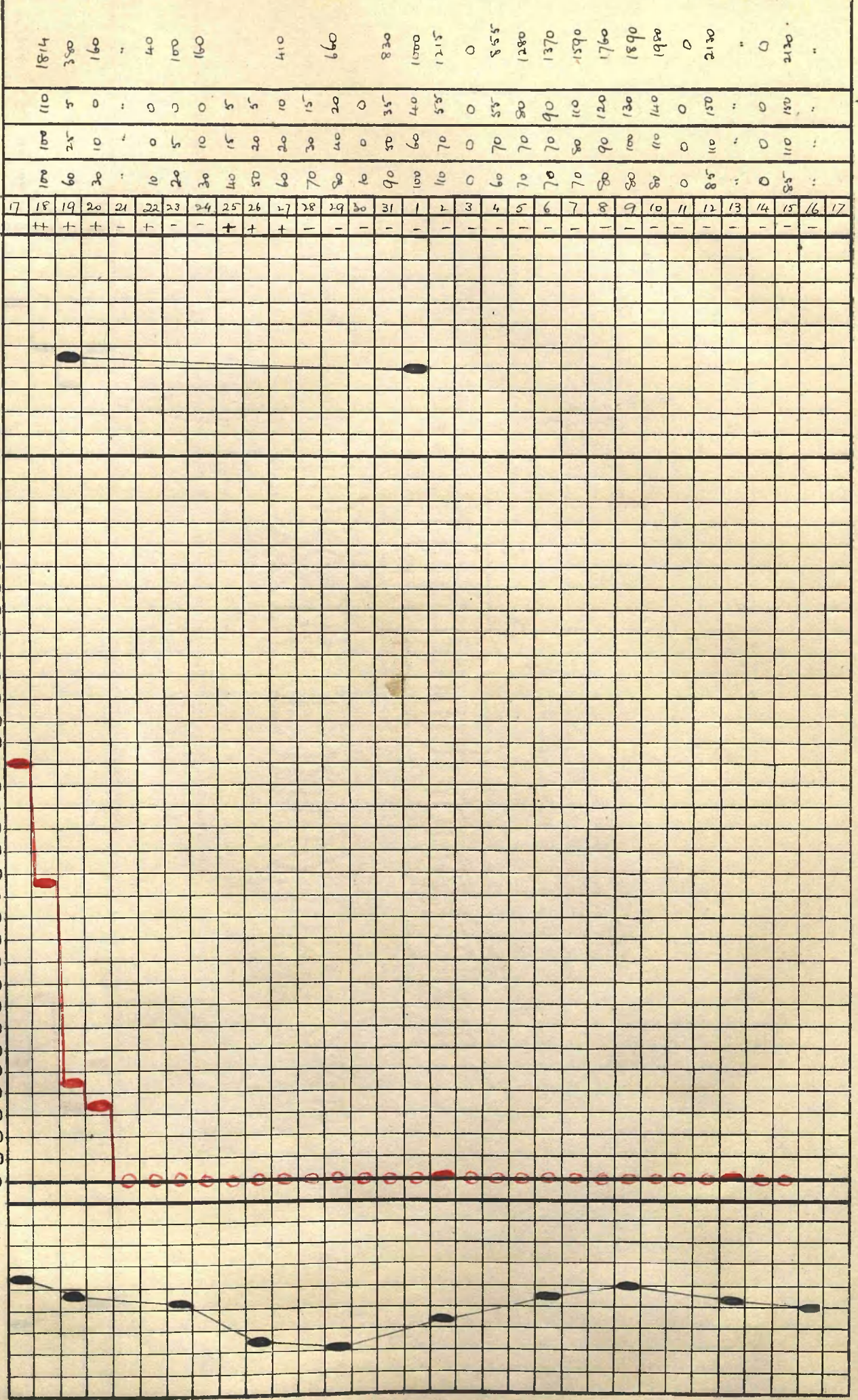
Name V.S. Date March 17  
 Age 31 ACETONE  
 Hard 0.45 420  
 Urinal 0.40 410  
 Page 0.35 400  
0.30 390  
 BLOOD 0.25 380  
 SUGAR 0.20 370  
 PER 0.15 360  
 CENT 0.10 350  
0.05 340

TOTAL URINARY SUGAR

Date of Admission... 11.3.12

INSULIN UNITS.

WEIGHT IN KILOS









Vernon  
Gray

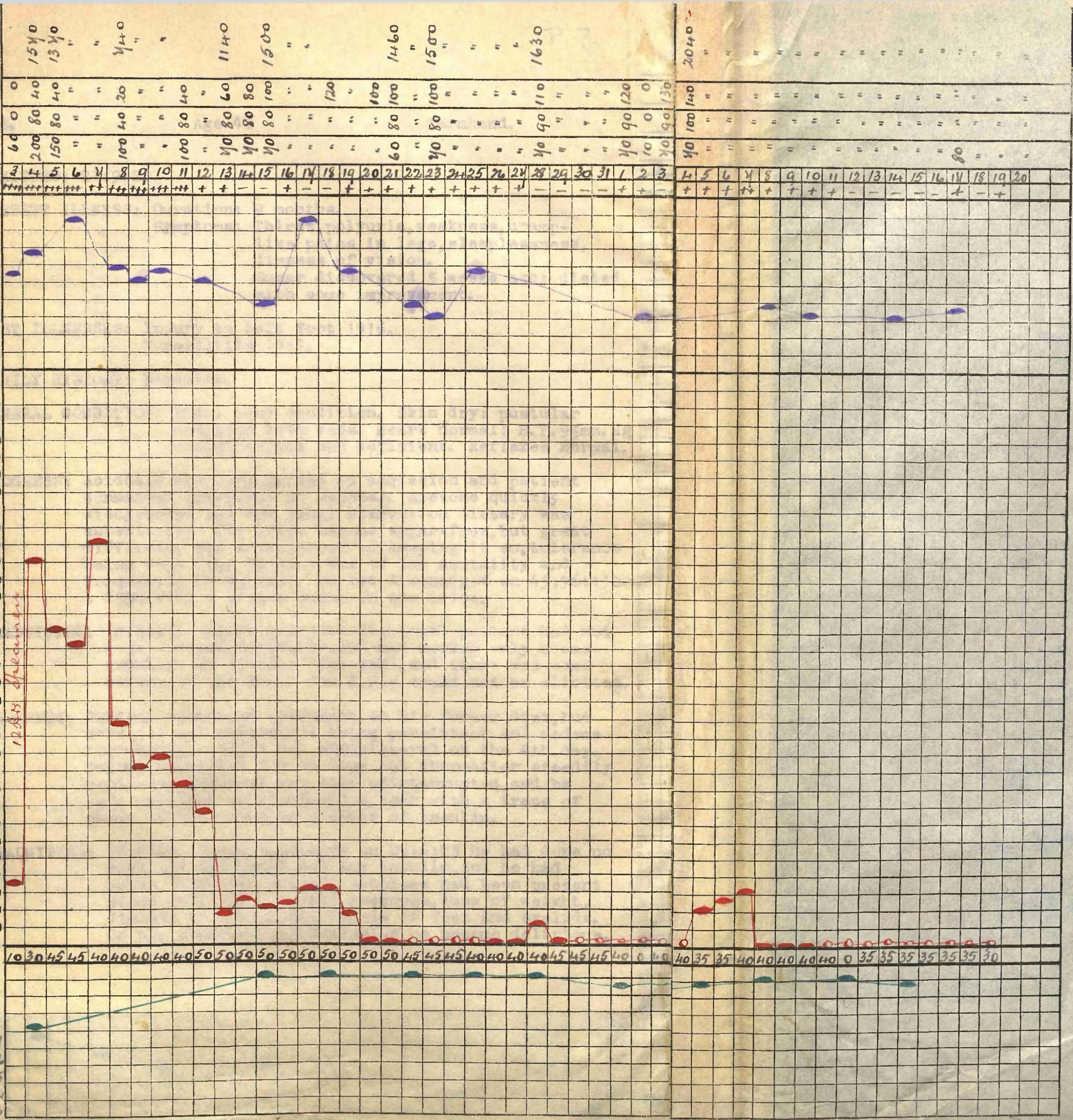
Age... 0.45  
Ward... 0.40  
Journal... 0.35  
Page... 0.30  
BLOOD SUGAR PER CENT  
0.25  
0.20  
0.15  
0.10  
0.05

TOTAL URINARY SUGAR

Date of Admission... 3.10.23

INSULIN UNITS.

WEIGHT IN KILOS





R. S. Age 42 Farmhand.

ADMITTED: 21:9:22.

PRESENT ILLNESS: Duration: 9 months.

Symptoms: Thirst, polyuria, weakness, cramp-like pains in legs, sleeplessness, dimness of vision.

Sugar discovered 5 weeks ago: dieted with some improvement.

PAST ILLNESSES: Injury to Left foot 1916.

Tonsillitis 1917.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin: poor condition. Skin dry: pustular eruption both legs. Heart normal: B.P. 95mm.Hg. Teeth carious and deficient. Reflexes normal.

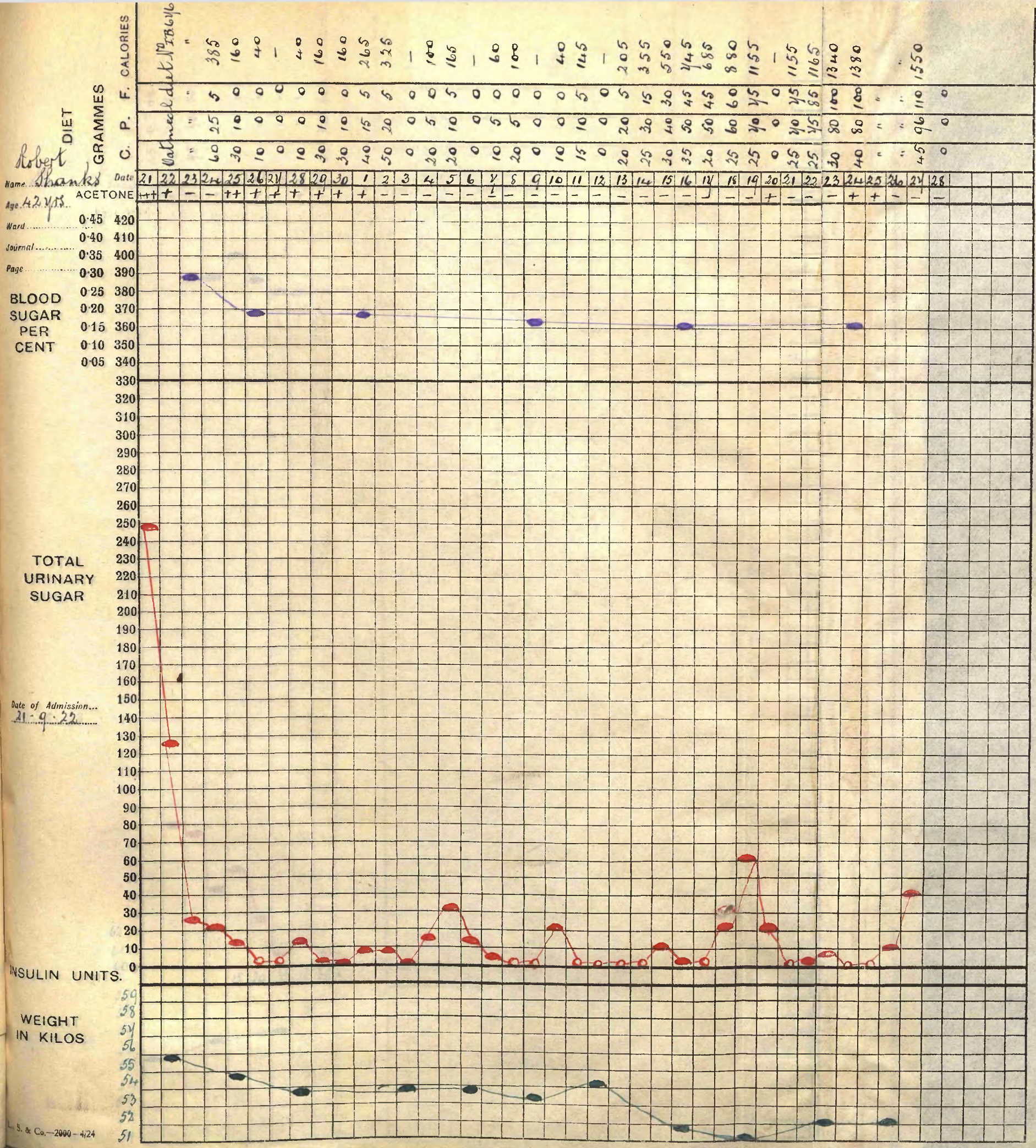
PROGRESS: Acidosis was very marked on admission and patient commenced treatment on Oatmeal. Acetone quickly disappeared and the usual starvation dietary was substituted. The urine became sugar-free, but great difficulty was experienced in keeping it so, tolerance being very low. Patient was of low mentality and frequently broke diet. He was discharged on 45:96:110 = 1550, with a little sugar in the urine.

READMITTED: 12:5:23. Since previous dismissal patient had not been dieting carefully, and had worked only occasionally. He did not look well, and there was some oedema of the feet. The K.J.s could not be elicited.

PROGRESS: Patient commenced treatment on his former diet, but acidosis and glycosuria being persistent and oedema increasing insulin was administered on the 4th. day. Oedema increased for 10 days but thereafter steadily declined. Progress was then uninterrupted, and he left hospital on 75:85:100 = 1540, with a trace of sugar in the urine on 5 units of insulin.

READMITTED: 17:4:24. Since discharge on 25:6:23 he had done no work. He had never had any insulin, and he had eaten 'what was going'. Symptoms had been present since Jan. 1924 - marked weakness, loss of weight, dimness of vision, and oedema of feet and eyelids. On the night before admission he became delirious.











J.D. 1923: Patient born Age 5 treatment of Schoolboy.

ADMITTED: 7:9:22. decreased diet and increased insulin

PRESENT ILLNESS: Duration: 2½ months. Symptoms: Thirst, polyuria, loss of weight, weakness, constipation. = 1340, with 25 units of insulin, free from symptoms.

PAST ILLNESSES: Scarlet Fever 1918  
Jaundice 1922.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin. Skin moist. Heart and lungs normal.  
Tongue dry. Reflexes normal.

PROGRESS: On a somewhat restricted diet symptoms quickly disappeared, and patient soon returned to full diet. Glycosuria was now intermittent and never present in amount. The blood sugar never rose above normal limits save on one occasion. A Glucose Test gave a 'Lag' curve. He left hospital free from symptoms.

READMITTED: 20:6:23. Patient had been very well until May 1923 when thirst and polyuria returned. He was admitted for insulin treatment.

This case was of little significance, but later admissions showed the true nature of

PROGRESS: Patient was put upon his previous diet and continued on this with slight increase for 5 weeks. Although glycosuria was minimal or absent weight was steadily lost. Insulin was then administered with little benefit: but on 23:8:23 the diet was more than doubled with increased doses of insulin. The boy's condition improved steadily, weight increasing and glycosuria being readily controlled. As insulin could not be obtained for him at home its use was stopped before discharge. Sugar at once returned to the urine and weight began to fall. He left hospital on 30:8:23 = 1720. A Glucose Test on 21:7:23 gave now a typical Diabetic curve.

READMITTED: 7:6:24. Fairly well since discharge on 12:10:23. He had been examined periodically and recently had shown a steady deterioration with return of symptoms. Physical examination showed no change in condition.



















Mrs McM.

Age 33

Housewife.

ADMITTED: 20:7:20.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Thirst, polyuria, frequent attacks of vomiting.

Sugar discovered 1 month ago.

PAST ILLNESSES: Measles in childhood.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished. Good colour. Skin moist.  
Heart and lungs normal. Tongue coated.  
Reflexes normal.

PROGRESS: Patient was treated first on light diet, then starvation, then oatmeal. Starvation following this cleared the urine of sugar. The diet was now rapidly increased but glycosuria did not recur. The patient left hospital on  
90: 150: 50 = 1410, free from symptoms.

READMITTED: 7:10:20. Patient was very well, but she was unable to control the glycosuria which had now reached 12.5%. She was readmitted for training.

PROGRESS: Patient was treated on the usual dietetic lines and sugar rapidly disappeared from the urine. Progress thereafter was uninterrupted, the diet on dismissal on this occasion reaching  
130: 130: 120 = 2120, without return of symptoms.

Patient was seen from time to time during the following six months. The glycosuria was never more than a trace and the weight was well maintained.

READMITTED: 28:4:24. Patient had been very well until two months before when persistent glycosuria had induced her own doctor to administer insulin cautiously, but in spite of this the urine did not become sugar-free and other symptoms (thirst, polyuria) had returned.



She was admitted to regulate the insulin dosage. The skin was very dry, but the general condition otherwise was unchanged.

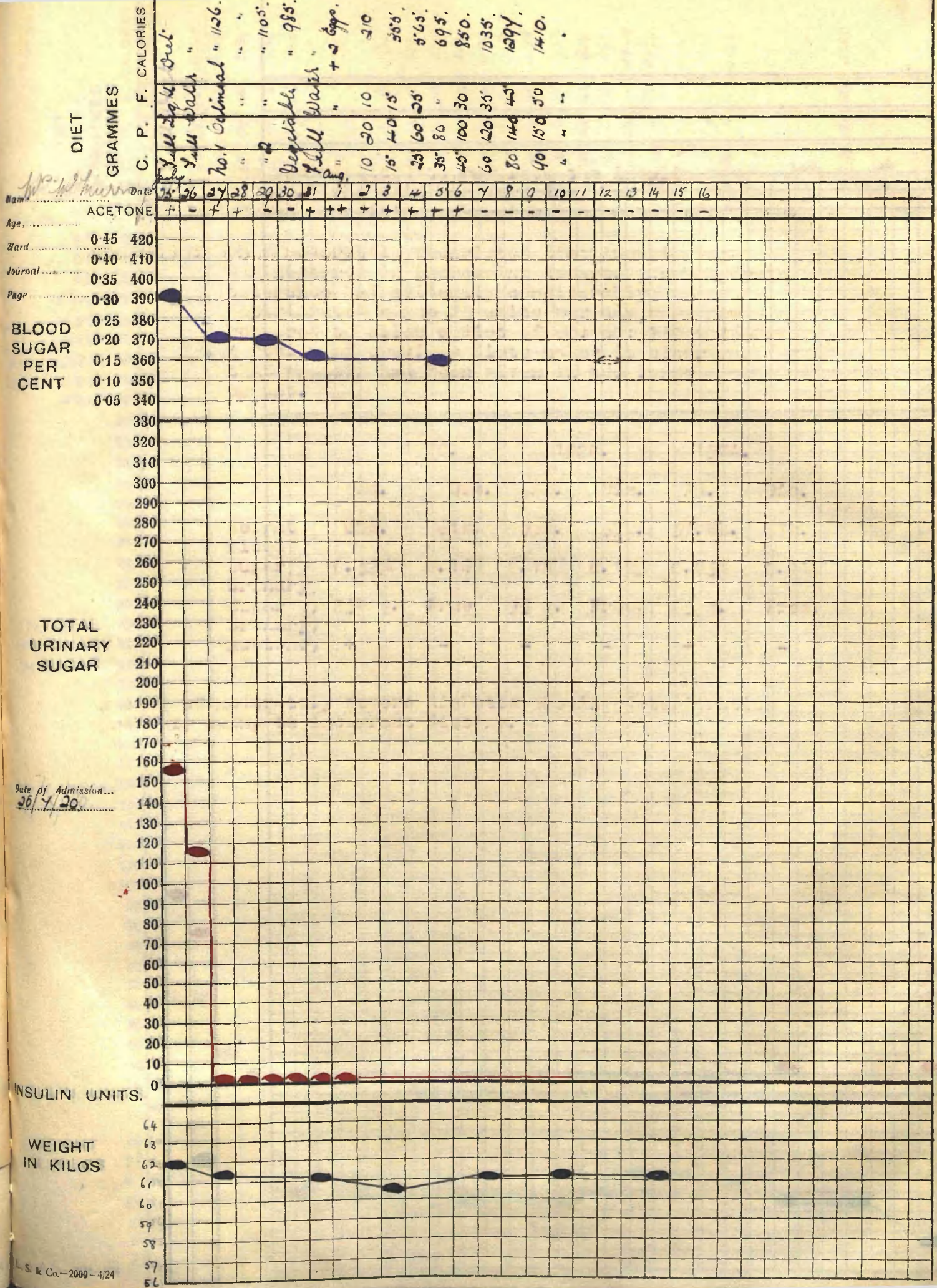
**PROGRESS:**

On a moderately restricted diet insulin was administered in increasing dosage. Carbohydrate tolerance had evidently considerably deteriorated since 25 units per day were now required to allow a diet of 60: 60: 130 = 1650. A review of previous diets makes it clear that protein had been given in too large amount.

	1920.		1920.		1924.	
	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.
Weight	62k.	61k.	59k.	59k.	57.8k.	57k.
Blood)						
Sugar)	0.32%	0.16%	0.18%	0.12%	0.25%	0.11%
Urinary)						
Sugar )	256 g.	Free	95 g.	Free.	85 g.	Free.
Urinary)						
Acetone)	+	-	-	-	+	-

A case of moderately severe diabetes shewing deterioration probably owing to incorrect dieting.







DIE T

GRAMMES

C P F CALORIES

Full	Buck
" "	" "
100	110
63	54
13	6
Full Buck	Dick.
" "	" "
" "	" "
13	6
25	10
40	30
45	50
45	70
50	85
60	95
80	110
90	120
100	"
110	130
120	"
"	"
"	"
30	"
120	130
120	120
120	120

7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1
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[illegible]

Year	Number of people (millions)
1950	15
1960	25
1970	30
1980	32
1990	35
2000	45

120	110	110
-----	-----	-----

10	


[illegible][illegible]

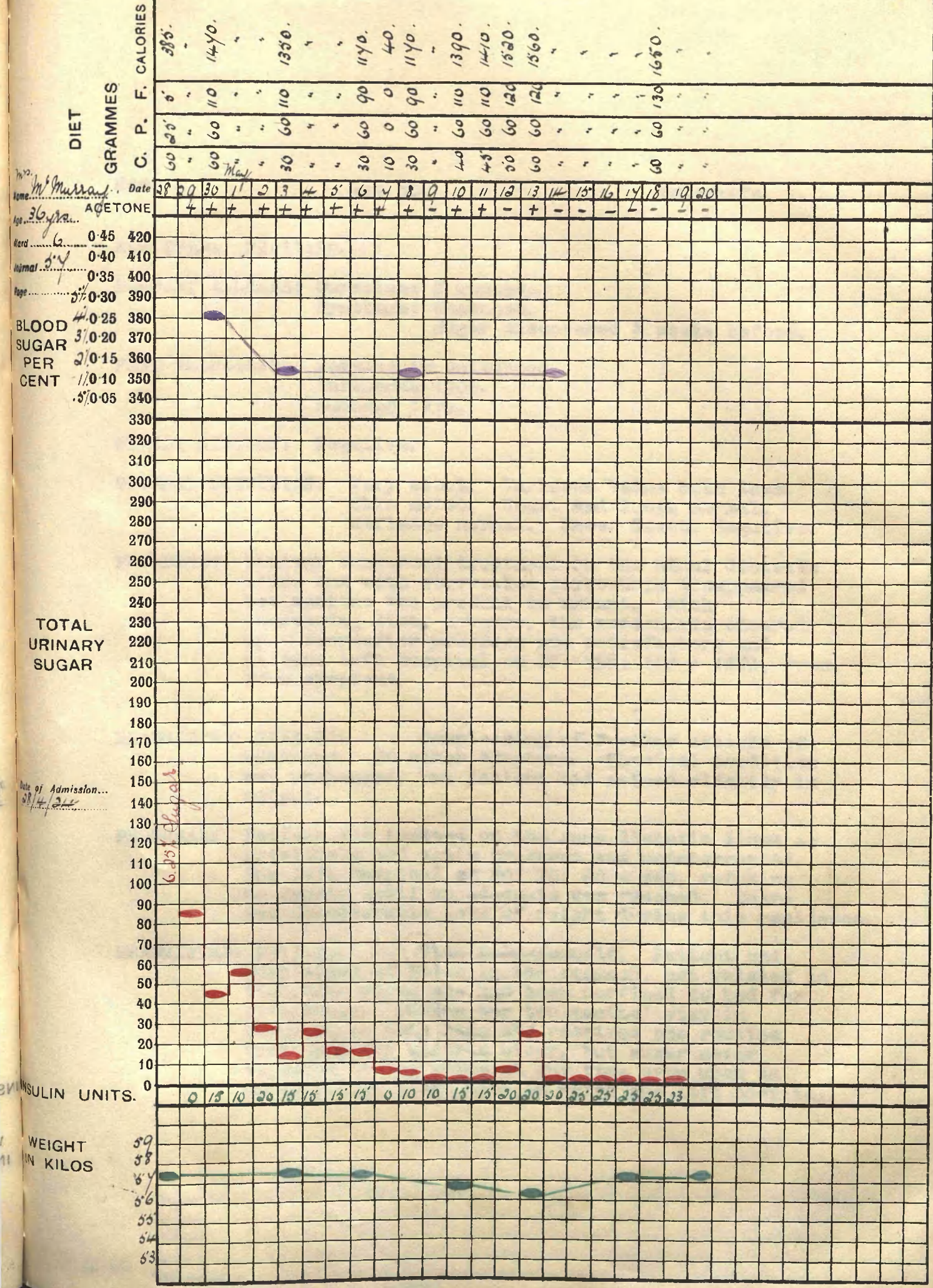
"			
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[illegible][illegible]

A full-page view of a sheet of graph paper. The paper has a light cream or off-white background. A dark grid of thin lines covers most of the page, forming small squares. On the left side, there is a vertical strip of yellowed or discolored paper. In the bottom-left corner, there are some faint, handwritten red markings that appear to be numbers like "3/4" and "1/2". The right edge of the paper shows a slight shadow, suggesting it's part of a bound notebook.

Column	Row
0	5
1	3
2	2.5
3	3.5
4	3.5







Mrs. M.

Age 50.

Housewife.

ADMITTED: 22:11:20.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Weakness.

Sugar discovered 4 weeks before.

PAST ILLNESSES: Measles in childhood.

Influenza 1906.

Lumbago 1919.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Very stout. Varicose Veins both legs.  
 Skin moist. Heart and lungs normal.  
 Reflexes normal. Wass. React. Negative.

PROGRESS: Patient commenced treatment on the usual dietetic lines and with starvation glycosuria disappeared but acetone was present in amount. With increasing diet, however, the acetonuria cleared up. Thereafter progress was satisfactory and patient left hospital on 80: 120: 120 = 1860, free from symptoms.

READMITTED: 21:5:21. Complaining of further attacks of weakness. No other symptoms. Physical condition was unchanged, but patient had gained slightly in weight.

PROGREAA: Patient was treated on the same dietetic lines as previously and again progress was uninterrupted. She left hospital on 80: 70: 40 = 960, refusing to remain until an adequate was reached. There was considerable loss of weight during this residence.

READMITTED: 23:7:23. With Haematemesis. Patient had complained of pains in the stomach, not related to food, with which she had been confined to bed for five weeks. During her two months' stay in hospital at this time she received the routine treatment for gastric ulcer, but sugar never appeared in the urine at any time even upon an unrestricted light diet on which she left hospital.



DIET

GRAMMES

DATE

ACETONE

BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

INSULIN UNITS

WEIGHT IN KILOS

	1920.		1921.		1923.	
	Ad.	Dis.	Ad.	Dis.	Ad.	Dis.
Weight	88k.	88k.	89k.	83k.	-	80k.
Blood Sugar	0.22%	0.19%	-	0.12%	-	-
Urinary Sugar	125 gms.	Free.	30 g.	Free.	Free.	Free.
Urinary Acetone	-	-	-	-	-	-

A case of mild diabetes satisfactorily treated by dieting. The prolonged period of undernutrition necessitated by the haematemises following gastric ulcer would appear to have exerted a very beneficial effect in raising the carbohydrate tolerance.











Mrs. McN.

Age 62.

Housewife.

ADMITTED: 1:5:22.

PRESENT ILLNESS: Duration: 6 years.

Symptoms: Thirst, polyuria, pruritis vulvae. Dieted occasionally during six years, latterly complaining of debility and nervousness.

PAST ILLNESSES: None.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Rather stout. Skin moist. Varicose Veins both legs. Slight oedema of feet. Arteries sclerosed. Reflexes normal. Wass. React. negative.

PROGRESS: Patient was treated on the usual dietetic lines and sugar rapidly disappeared from the urine. Subsequent progress was fairly satisfactory though some difficulty was experienced in reaching a reasonable diet. Patient developed a sore on the left heel which healed slowly. She left hospital on 50: 70: 90 = 1290, with a trace of sugar in the urine, but generally much improved.

READMITTED: 16: 4: 23. Complaining of pains in the legs and feet and numerous ulcers on the toes. She stated she had lost weight.

GENERAL CONDITION: Little changed, but the oedema now extended on to the legs and trunk and there were numerous rhonchi in the chest. The K.Js. were +. Numerous ulcers on the toes of the right foot: tip of right great toe gangrenous.

PROGRESS: Patient was treated on an equilibrium diet and glycosuria throughout was minimal. The blood sugar always ran high but acidosis gave rise to no anxiety. The ulcers progressed slowly towards healing and there was no spread of the gangrene. A few doses of insulin (5 units) were administered but the supply was insufficient at that time to allow of its continued use. Patient left



hospital on 80: 70: 90 = 1410, slightly improved. There was albuminuria throughout.

		1922.		1923.	
		Ad.	Dis.	Ad.	Dis.
Weight		59.2k.	56.2k.	53.2k.	52.2k.
Blood Sugar		-	B. 23%	0.25%	0.28%.
Urinary Sugar	35 gms.	6 gms.	20 gms.	Trace.	
Urinary Acetone	-	-	-	-	-

A case of glycosuria in a "done" old woman with arteriosclerosis.

TOTAL  
URINARY  
SUGAR

UNITS.



*Mr. McNeill*

Name *McNeill* Date *1/2*

Age *45*  
Ward *420*  
Journl *410*  
Page *400*  
*0.30* 390

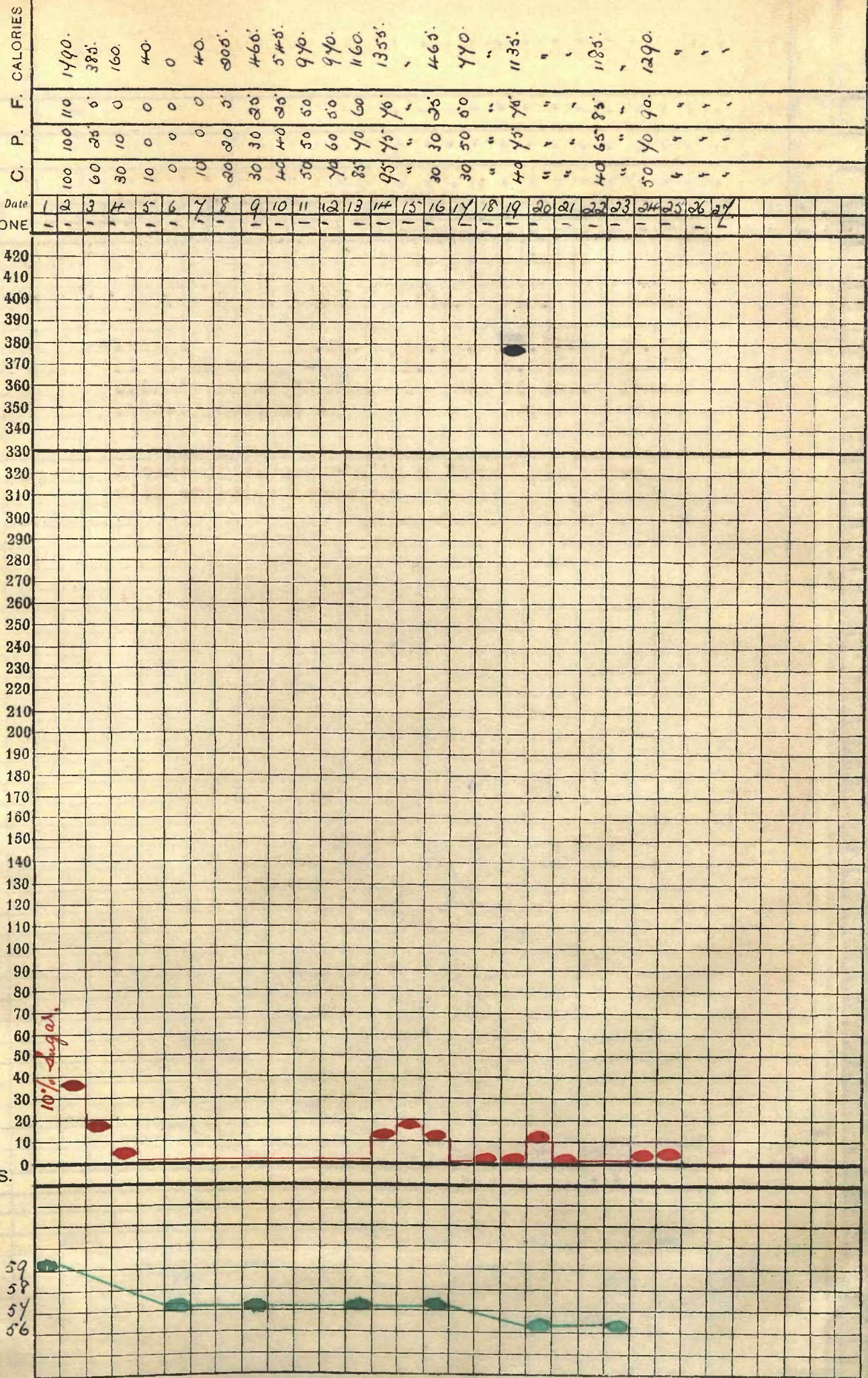
BLOOD SUGAR PER CENT  
*0.25* 380  
*0.20* 370  
*0.15* 360  
*0.10* 350  
*0.05* 340

TOTAL URINARY SUGAR

Date of Admission... *1/5/22*

INSULIN UNITS.

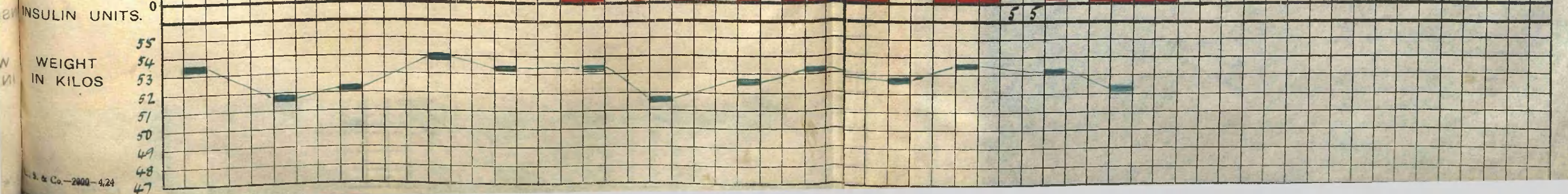
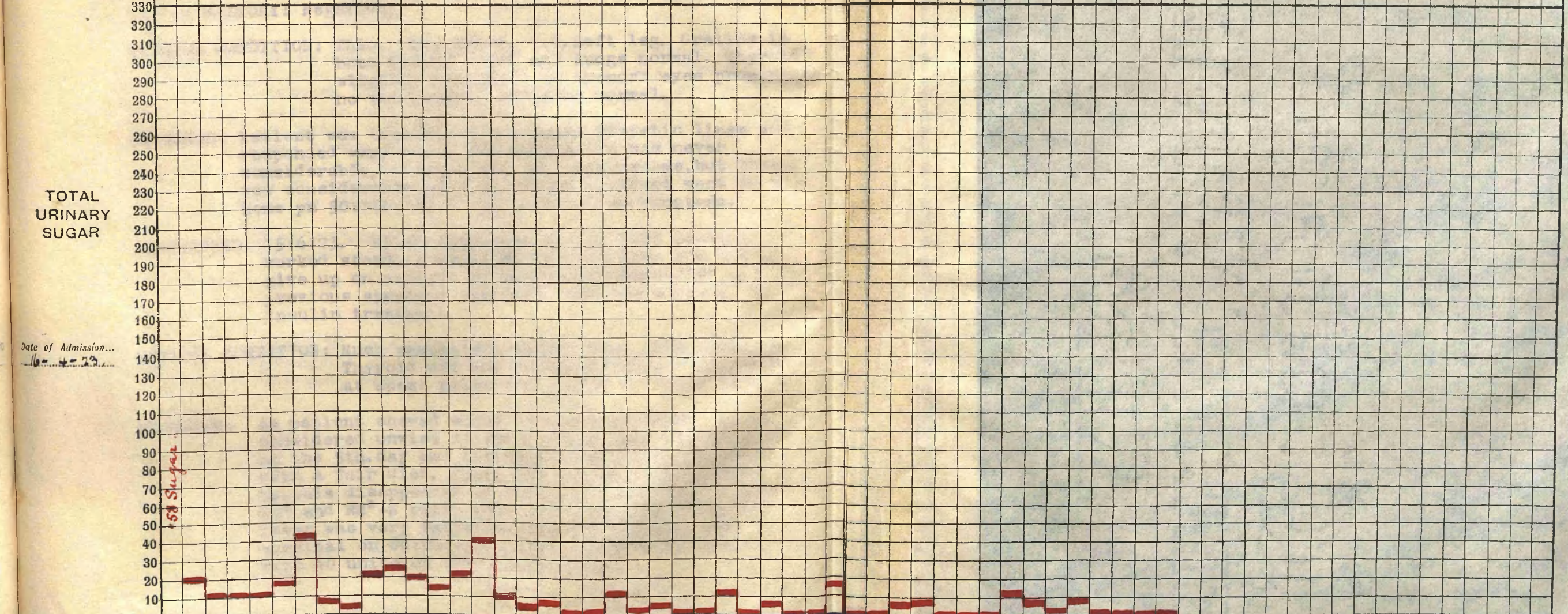
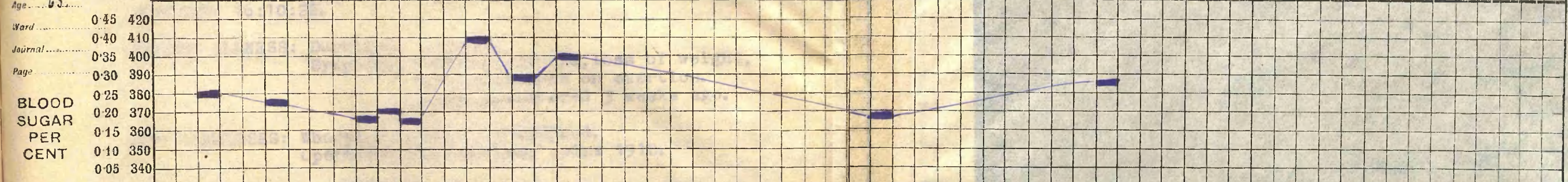
WEIGHT IN KILOS





DIET		GRAMMES		C.		P.		F.		CALORIES	
		60		70		80		90		1240	
		60		70		80		90		1410	

Name: Mr. McNeill Date: 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2





M. McB.

Age 34

Housemaid.

ADMITTED: 16:10:22.

PRESENT ILLNESS: Duration: 3 weeks.

Symptoms: Thirst, polyuria, loss of weight,  
breathlessness on exertion.  
Sugar discovered 3 weeks ago.

PAST ILLNESSES: Whooping Cough in childhood.  
Operation for varicose veins 1910.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Thin. Skin moist. V.V. Left leg. Grating in both knees. Heart and lungs normal. Thyroid slightly enlarged: no tremor: eyes prominent: no von Graefe. Reflexes normal.

PROGRESS: Patient was treated on the usual dietetic lines and responded very well. The glycosuria was never considerable, acidosis was not troublesome, but there was considerable loss of weight. Patient went to the Home pn 50:110:140 = 1900, free from symptoms.

READMITTED: 15:6:23. After discharge on 14:11:22 patient worked steadily until April 1923 when she had to give up on account of weakness. Thereafter all her previous symptoms returned. She was admitted for insulin treatment.

GENERAL CONDITION: Much emaciated. Looked ill. Skin very dry. Thyroid not now enlarged. Short V.S. murmur at apex: pulse 100. Liver +. K.J.s absent.

PROGRESS: As patient shewed signs of cardiac failure it was considered unwise to starve her. Insulin was begun on the 5th. day and increased rapidly to 50 units with a fair diet. There was considerable oedema but this disappeared as the pulse rate fell. Alveolar  $\text{CO}_2$  and  $\text{NH}_3\text{-N}$  ratio were satisfactory. Progress later was very satisfactory, and patient left hospital on 80:90:150 = 2030, free from symptoms, with 40 units of insulin per day.



READMITTED: 6:2:24. Since discharge on 19:8:23 she had been working steadily as a cook. On 4:2:24 she complained of shortness of breath and pains 'all over', particularly in Rt. side of chest.

GENERAL CONDITION: Patient very ill. Considerable dyspnoea: respirations rapid and sighing ( air hunger ). No dulness to percussion but rale both bases. No oedema. K.J.s not elicited. Heart sounds poor: pulse rapid but of fair quality. Throat congested and dry: odour of acetone in breath. B.S. = 0.45%  
Alveolar air = 3.2%. Urine - Sugar 2.7%:  
Acetone +++ : Diacetic Acid ++.

PROGRESS: During the first 24 hours patient received 60 units of insulin, but on 7:2:24 she was very drowsy although the breath no longer smelt of acetone. The pulse ran about 140, poor in quality. B.S. = 0.414%. The urine was unchanged. On 8:2:24 patient was much less drowsy and the urine contained Acetone + : Diacetic Acid -. She was, however, very collapsed, the pulse being almost imperceptible. Cardiac failure became more extreme and patient died at 7 p.m.

Post Mortem Exam. Pancreas small and atrophied, the head most involved.

Bronchopneumonia.

Subacute Nephritis.

Death in this case was apparently due to Bronchopneumonia and Cardiac Failure rather than to diabetic coma.



Margaret  
Name *M. Burt*

Age *47*

Ward *4*

Journal *4*

Page *4*

BLOOD

SUGAR

PER

CENT

0.45 420

0.40 410

0.35 400

0.30 390

0.25 380

0.20 370

0.15 360

0.10 350

0.05 340

330

320

310

300

290

280

270

260

250

240

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210

200

190

180

170

160

150

140

130

120

110

100

90

80

70

60

50

40

30

20

10

0

INSULIN UNITS.

WEIGHT

IN KILOS

68

66

64

62

60

58

56

54

52

50

48

46

44

42

40

38

36

34

32

30

28

26

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ACETONE

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245







Margaret  
Unit Bld

Name Date

Age 45  
Ward 40  
Journal 35  
Page 30

BLOOD SUGAR PER CENT  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

TOTAL URINARY SUGAR

Date of Admission...  
4/11/24

INSULIN UNITS.

WEIGHT IN KILOS

385  
1140 (Apper)

P. B.

GRAMMES

DIET

C. 60  
P. 25  
F. 80  
E. 50  
CALORIES

ACETONE

12 Noon  
4 P.M.  
10 P.M.

60 30

2.4% Sugar  
1.1%

60 30



Miss P.

AGE 39

Hospital Nurse.

ADMITTED: 19:1:23.

PRESENT ILLNESS: Duration: 2 months.

Symptoms: Thirst, polyuria, defective vision.

Discovered sugar herself 1 month ago.

PAST ILLNESSES: Scarlet Fever in childhood.

Measles " "

FAMILY HISTORY: Negative.

GENERAL CONDITION: Well nourished. Fresh complexion. Skin healthy.  
 Odour of acetone in breath. Nothing abnormal  
 on physical examination.

PROGRESS: Patient was treated on the usual dietetic lines, but  
 tolerance was found to be low and glycosuria persisted  
 even on an inadequate diet. She left hospital to go on  
 holiday and to return for insulin treatment.

READMITTED: 21:4:23. Condition unchanged save that glycosuria  
 had increased.

PROGRESS: Patient went onto her previous dismissal diet and  
 insulin was commenced. The dose was quickly raised to  
 40 units with increased diet ( 90:80:120 = 1760 ) at  
 which she was free from symptoms. She developed an  
 abscess on the arm at the seat of injection which gave  
 little trouble. At her request insulin was stopped  
 before leaving hospital with a return of glycosuria.  
 Her diet on dismissal was 90:100:125 = 1885.

Jan.

April.

	Ad.	Dis.	Ad.	Dis.
Weight	50.5k.	48.2k.	51k.	52.2k.
Blood Sugar	0.10%	0.15%	0.36%	-
Urinary Sugar	45gms.	15gms.	68gms.	25gms.
" Acetone	-	-	-	-

A satisfactory result with insulin in a severe case.  
 Patient subsequently went onto 20 units of insulin  
 per day on which she was very well and fit for work.











A. P.

Age 15

Ward-maid.

ADMITTED: 5:2:23.

PRESENT ILLNESS: Duration: 4 months.

Symptoms: Thirst, polyuria, loss of weight,  
weakness, sleeplessness. No treatment.

PREVIOUS ILLNESSES: Whooping cough in childhood.

Measles " "

Erythema Nodosum 1922.

FAMILY HISTORY: Negative.

GENERAL CONDITION: Small: poorly nourished: anaemic. Skin dry.  
Heart normal. Lungs - dulness Lt. apex: V.F.  
and V.R.+: rale. Reflexes normalPROGRESS: On diet progress was rapid, sugar and acetone being  
quickly cleared from the urine. She was discharged  
on 45:55:105 = 1345, free from symptoms, and with  
the lung condition unchanged.Readmitted: 21:4:23. Since dismissal on 12:3:23 she had been  
at home. She had had a 'cold', and complained of  
weakness but of no other symptom. As her home  
circumstances were bad she was readmitted.  
Physical condition was unchanged save that the Lt.  
apex was more marked.Progress: Patient was put back onto her former diet, but as  
glycosuria was persistent insulin was administered  
and continued for 4 weeks. It was then thought  
that she was going to a Sanatorium and insulin  
was stopped but arrangements broke down at this  
time. Shortly after this the temperature began to  
rise in the evening and on 20:6:23 patient had a  
sharp haemoptysis. Haemoptysis recurred on 27:6:23.  
She was distinctly ill, and the lung condition was  
spreading. Insulin was resumed with increased diet  
and glycosuria was minimal. On 7:7:23 Tubercle  
bacilli were found in the sputum. On 6:7:23 patient  
was allowed full diet without sugar, and thereafter  
on this diet with a small dose of insulin the urine  
was generally sugar-free. No haemoptysis occurred  
after 3:8:23. Patient went to a Sanatorium on  
150:90:130 = 2130, with 5 units of insulin without  
sugar or acetone in the urine.

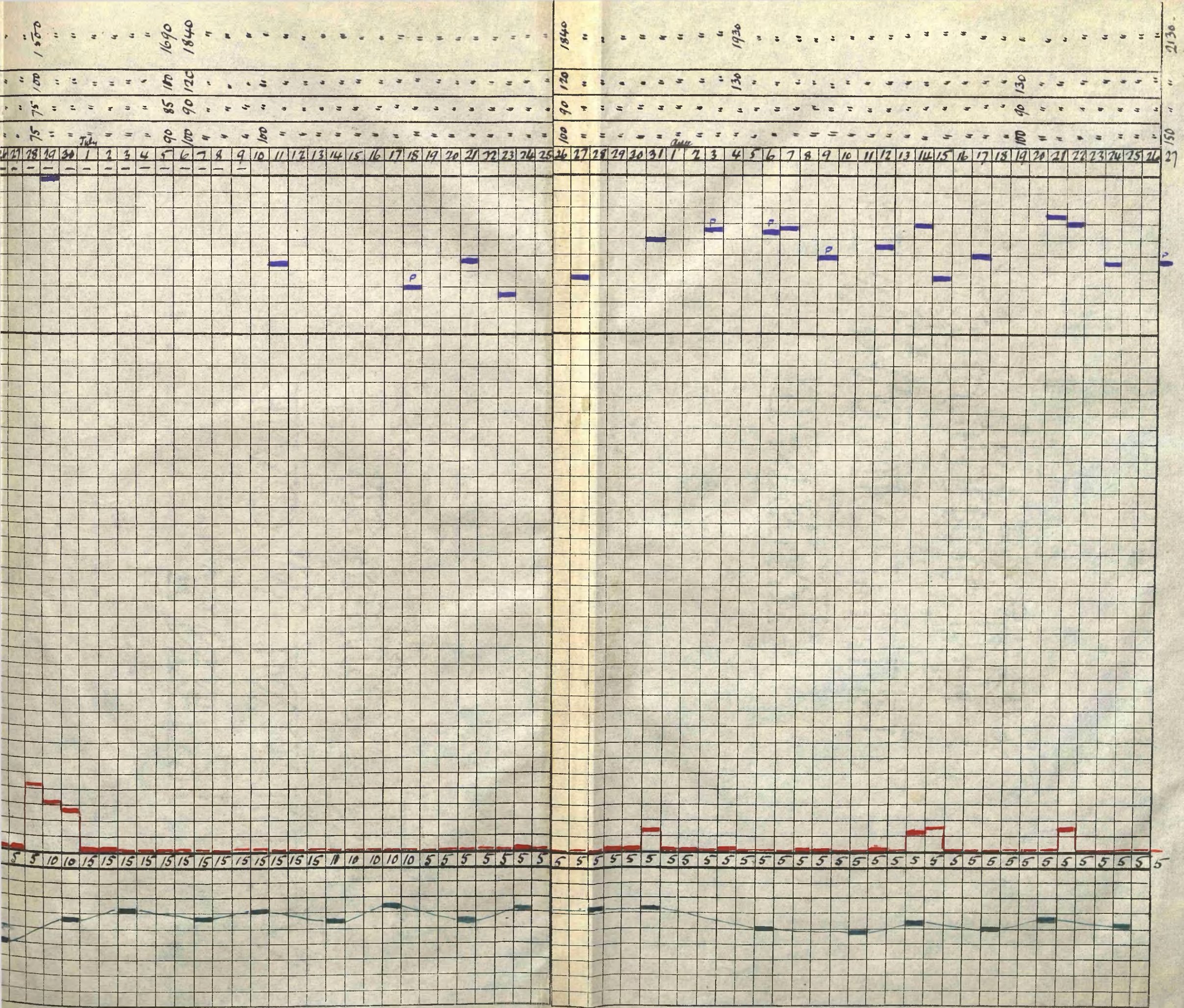














SECTION IV

(INSULIN)

CHARTS.



T. 1.

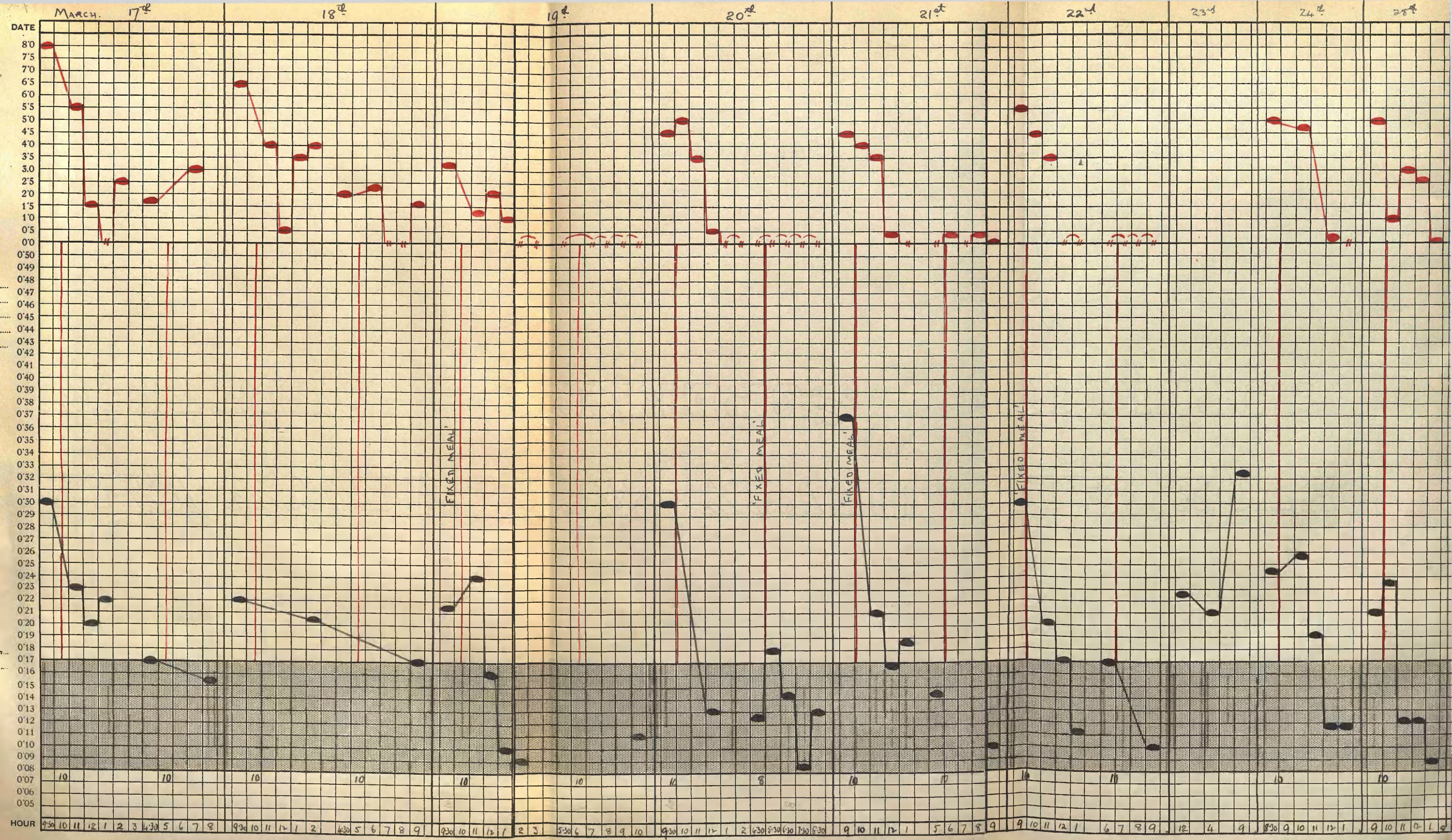
URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

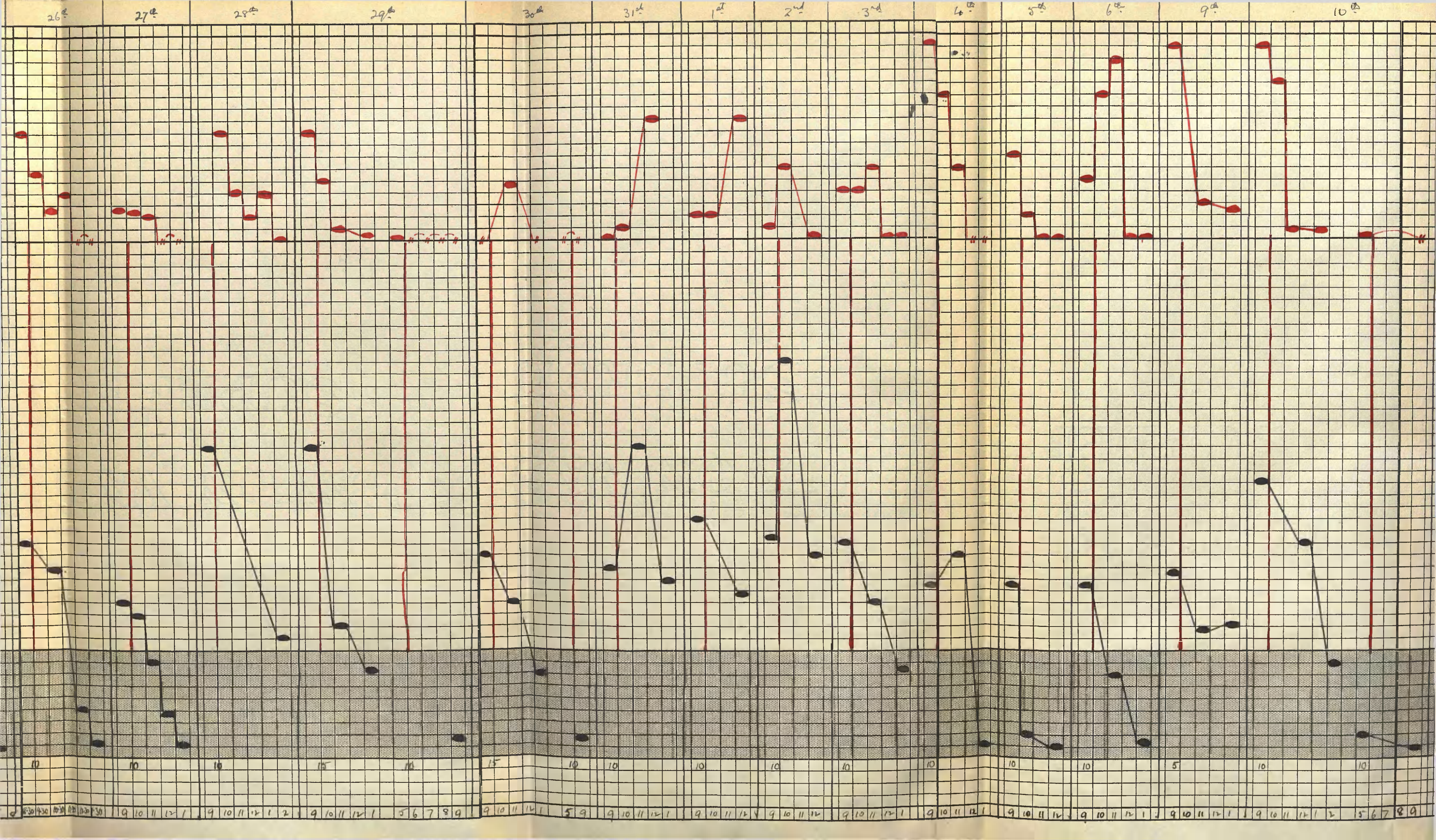
BLOOD  
SUGAR  
%

Date of Admission.....

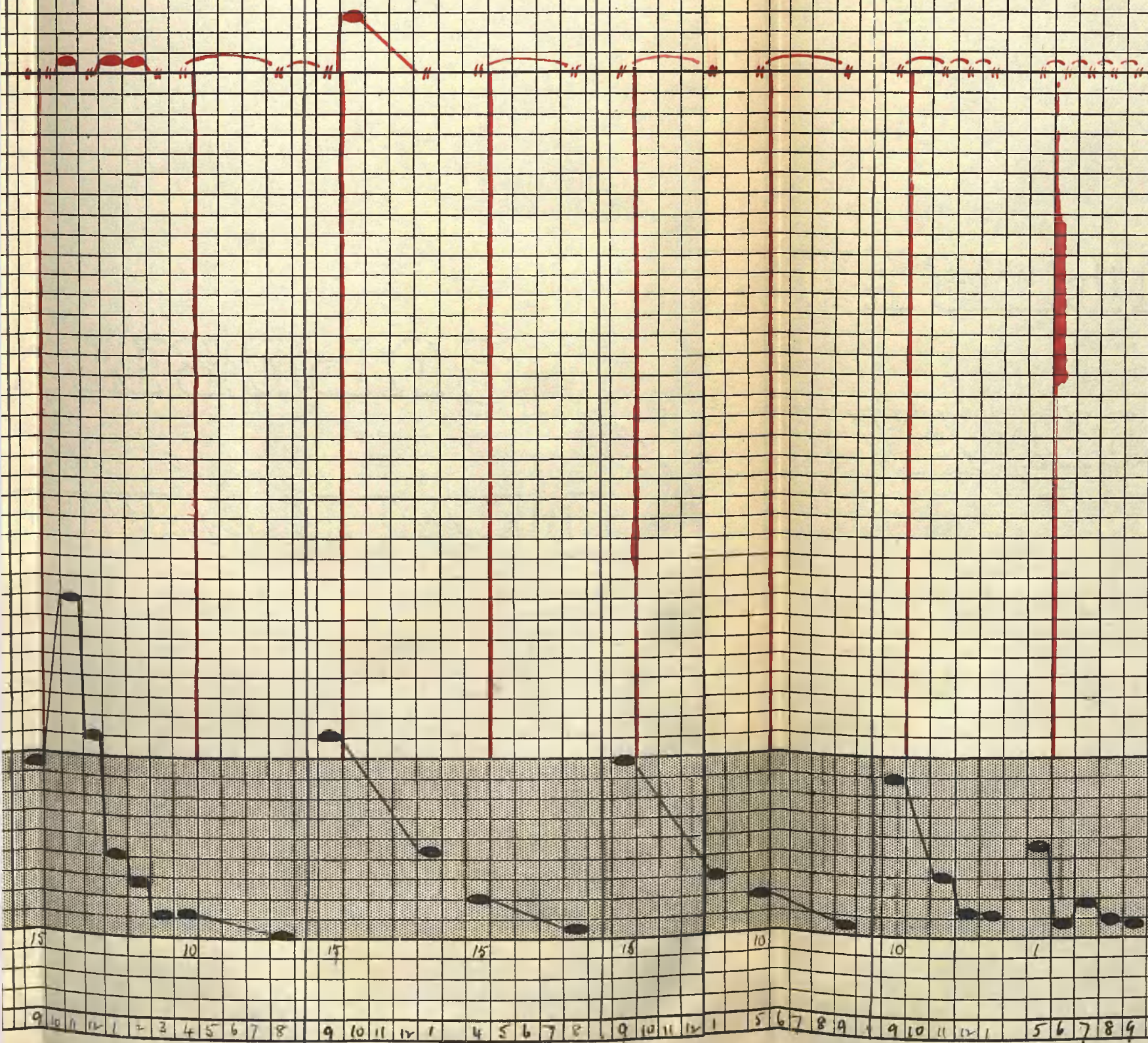
INSULIN  
UNITS.









11<sup>h</sup>12<sup>h</sup>13<sup>h</sup>14<sup>h</sup>



X

URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

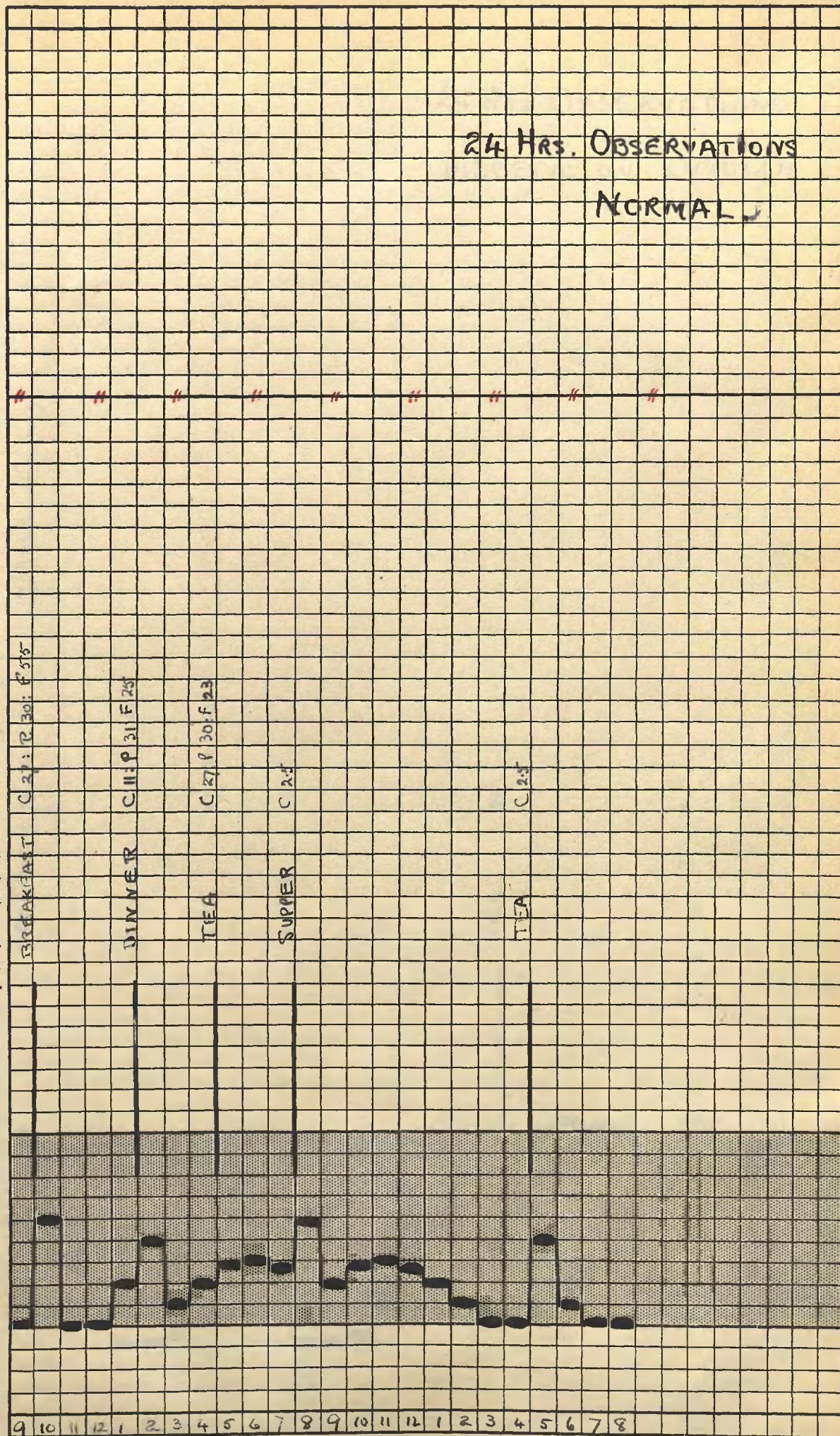
BLOOD  
SUGAR  
%

Date of Admission...  
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HOUR





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URINARY  
SUGAR  
%

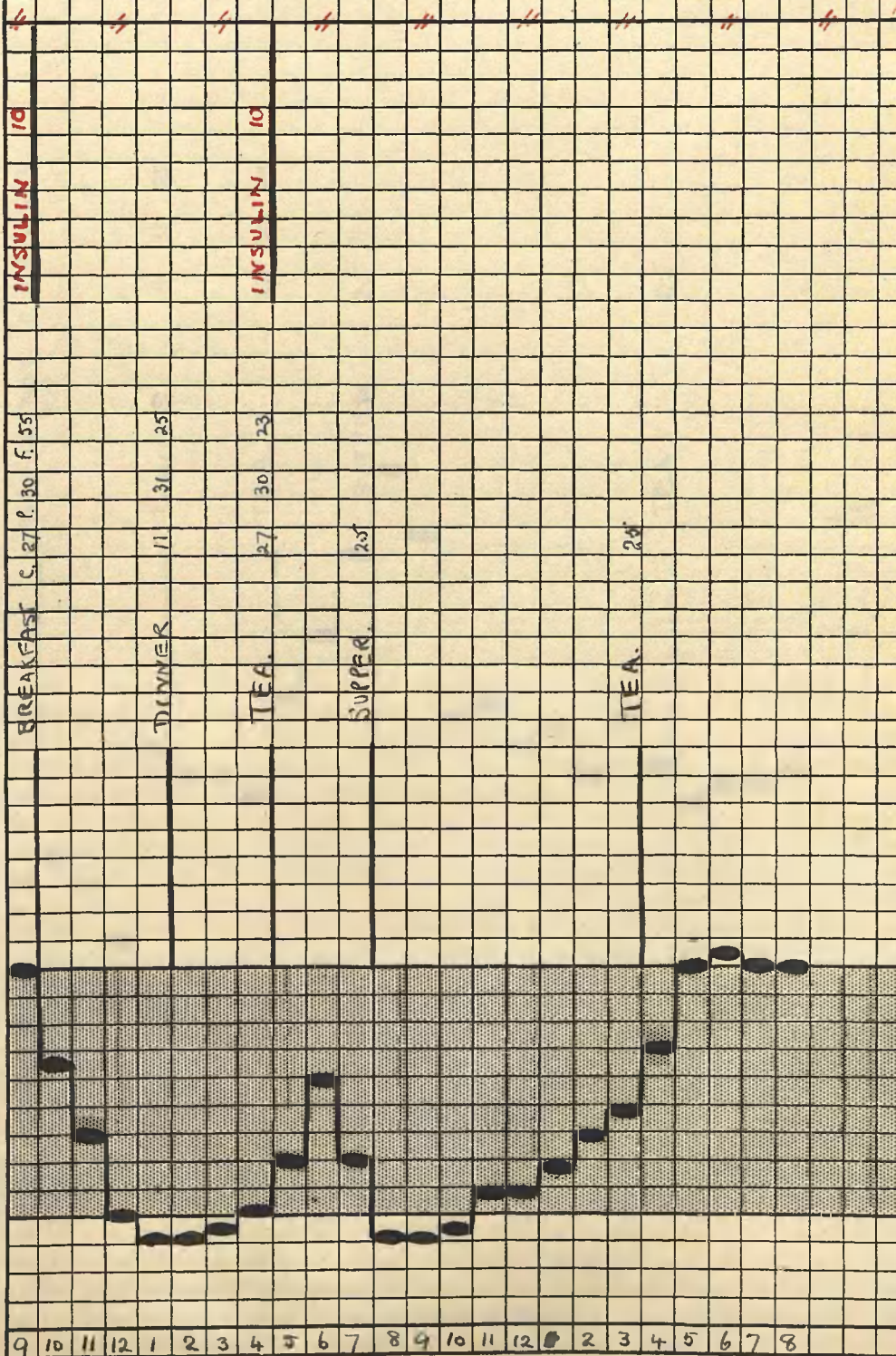
Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission...

24 Hrs. OBSERVATIONS

DIABETIC ON INSULIN





Z

URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission.....

DATE

80  
75  
70  
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HOUR

9 10 11 12 1 2 3 4 5 6 7 8 9 10 11 12 1 2 3 4 5 6 7 8

BREAKFAST C 27 P 30 F 58

DINNER

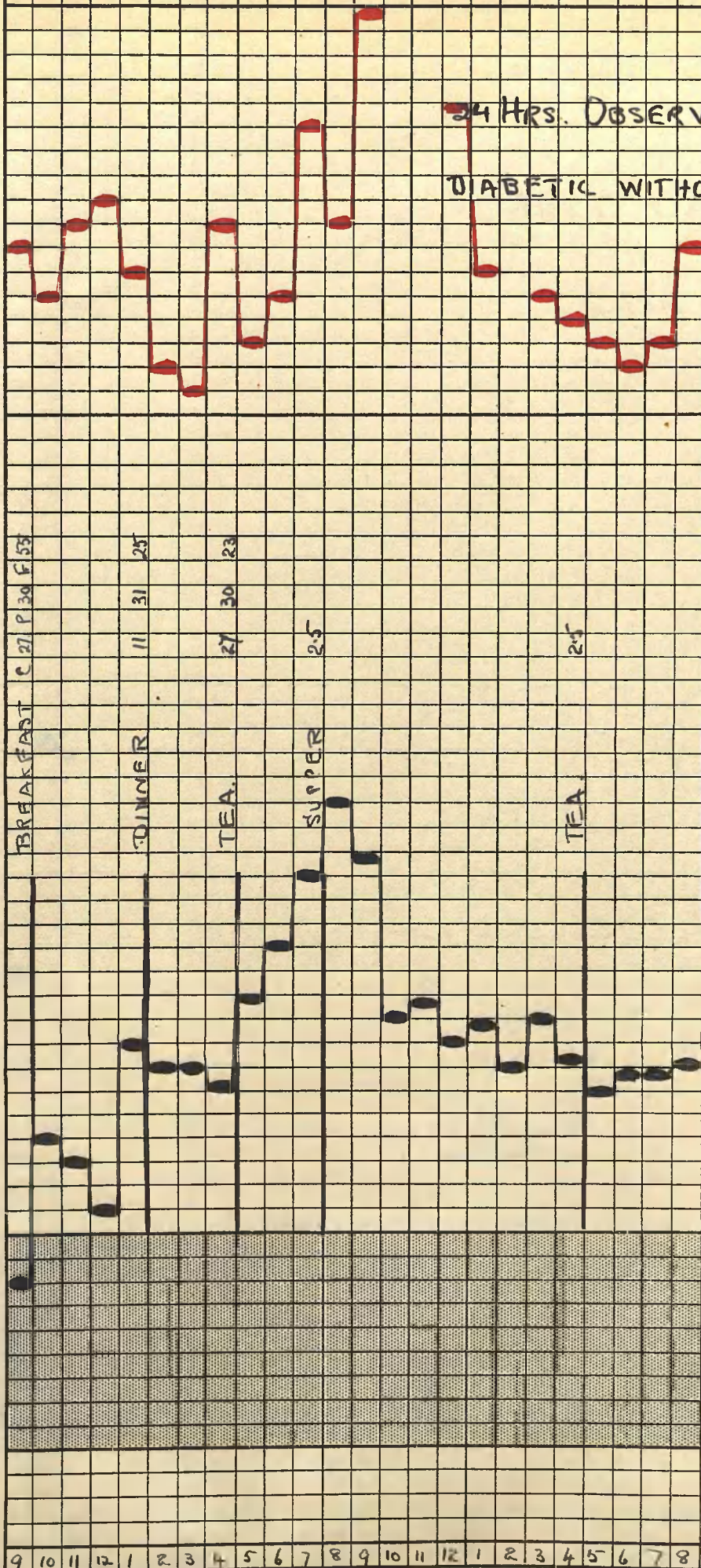
TEA

SUPPER 2.5

TEA 2.5

24 HRS. OBSERVATIONS

DIABETIC WITHOUT INSULIN.





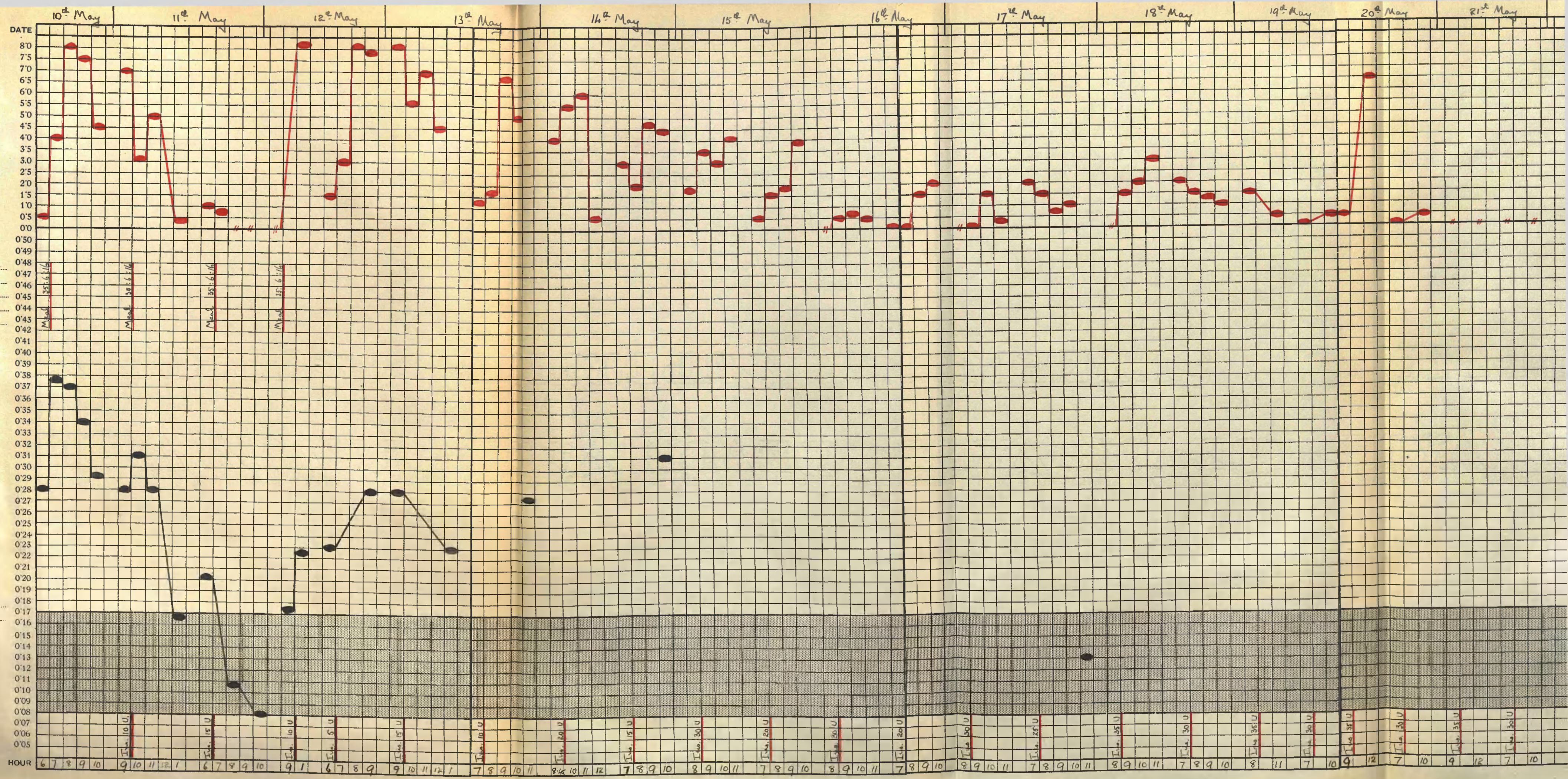
T. 2.

URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission.....





May

27 28 29 30 31 1 2 3 4 5 6 7 8 9

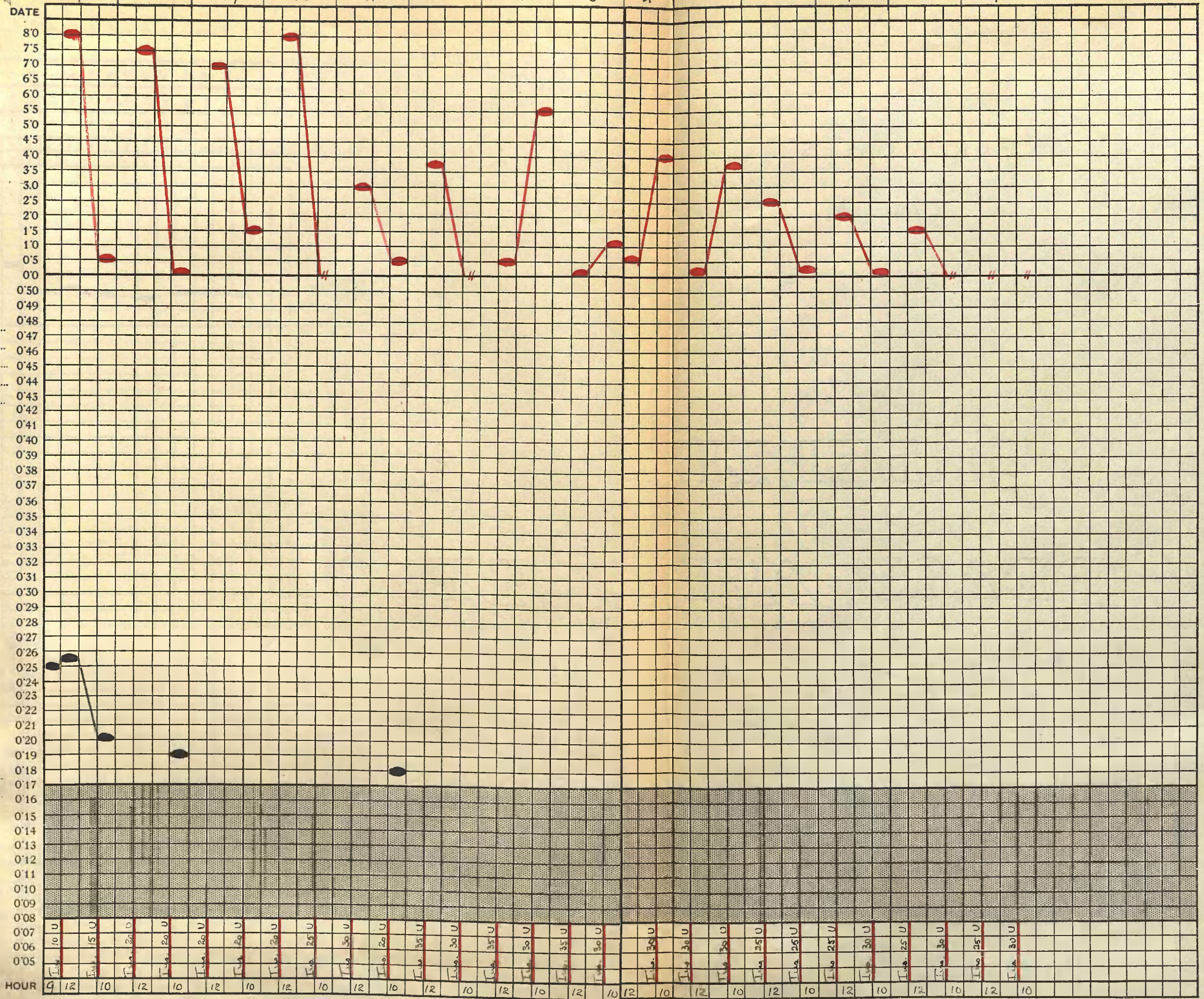
TA 3.

URINARY  
SUGAR  
%

Name *M. Neil*  
Age .....  
Ward .....  
Journal .....  
Page .....

BLOOD  
SUGAR  
%

Date of Admission...













U

DIET

GRAMMES

F. CALORIES  
P.  
C.

240	1570	1370		740		1080		1140	1320	1500		1680	1800	1460	1500
0	40			20		40		60	80	100		120	100	100	100
0	80			40		80		80	80	80		80	80	80	80
60	200	150		100		100		70	70	70		70	70	60	70

'ACID'

CASE

Date 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24

me. V.G. 32  
ACETONE  
0.45 420  
0.40 410  
0.35 400  
0.30 390  
0.25 380  
0.20 370  
0.15 360  
0.10 350  
0.05 340

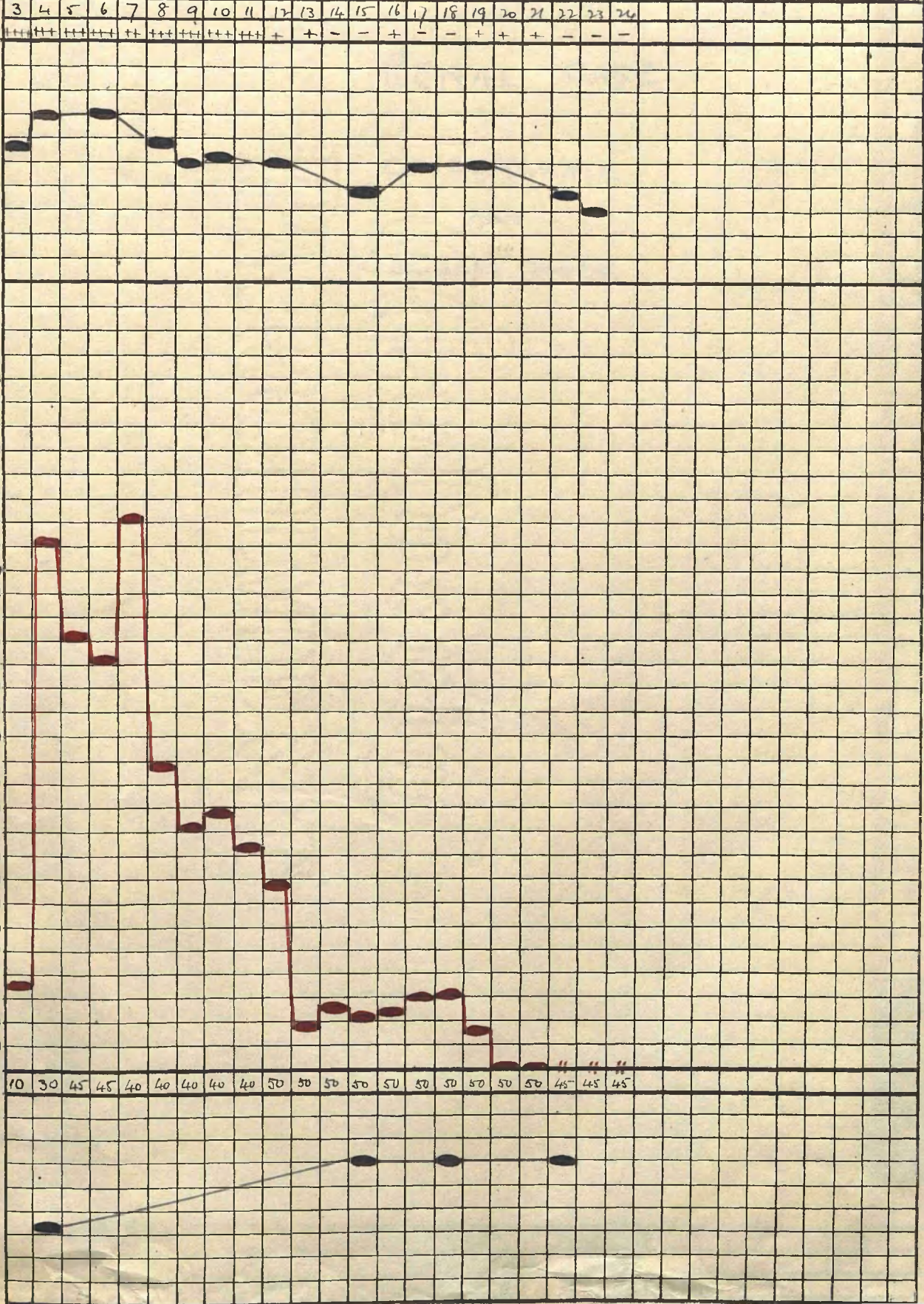
BLOOD SUGAR PER CENT

TOTAL URINARY SUGAR

Date of Admission... 3. X. 23.

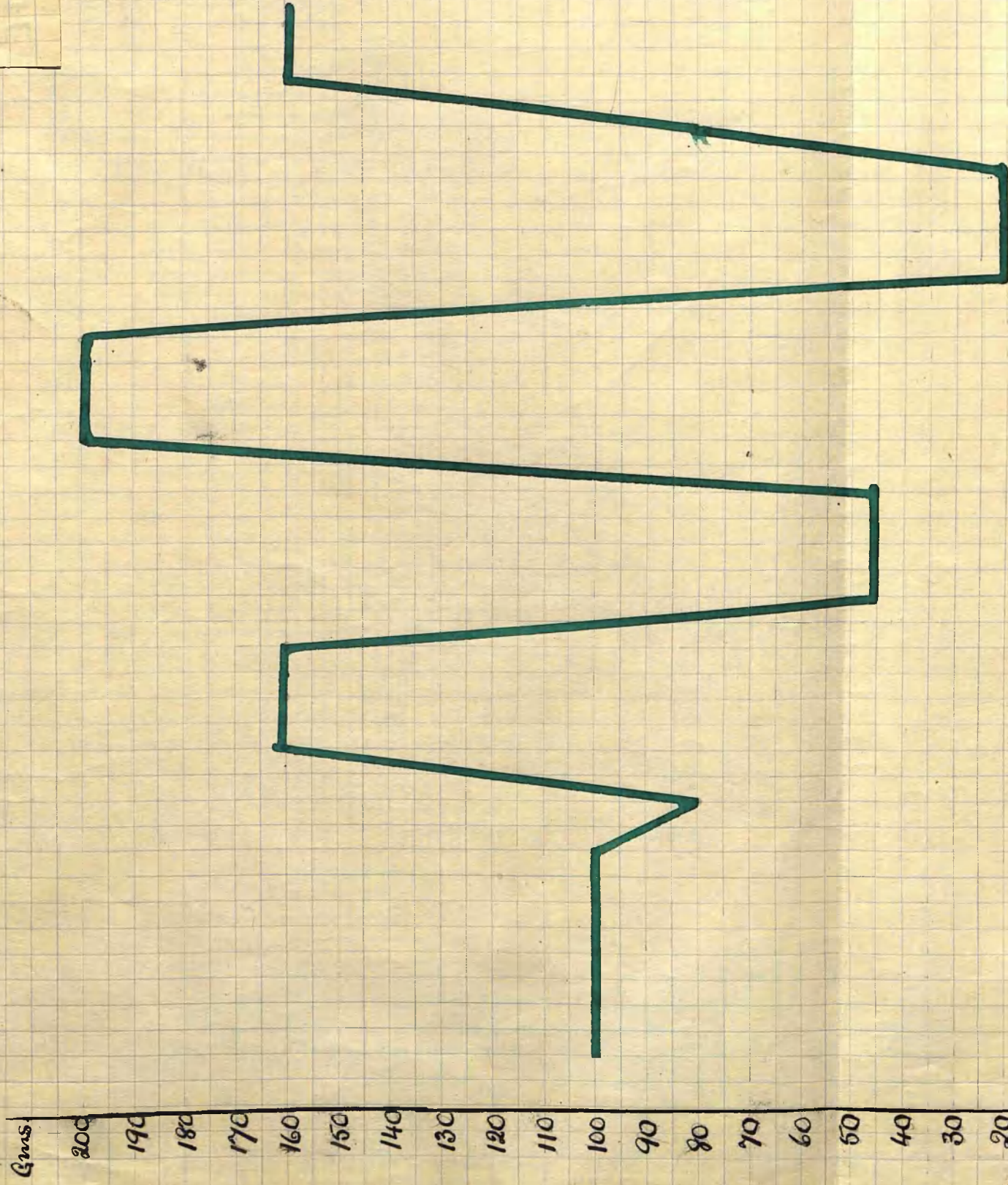
INSULIN UNITS.

WEIGHT IN KILOS





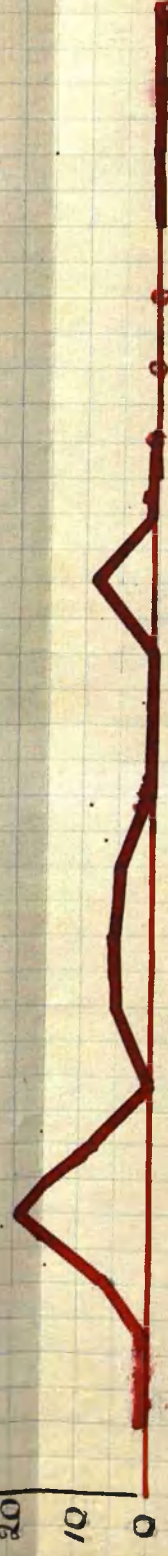
Mrs. NISBET.



Gms. 100

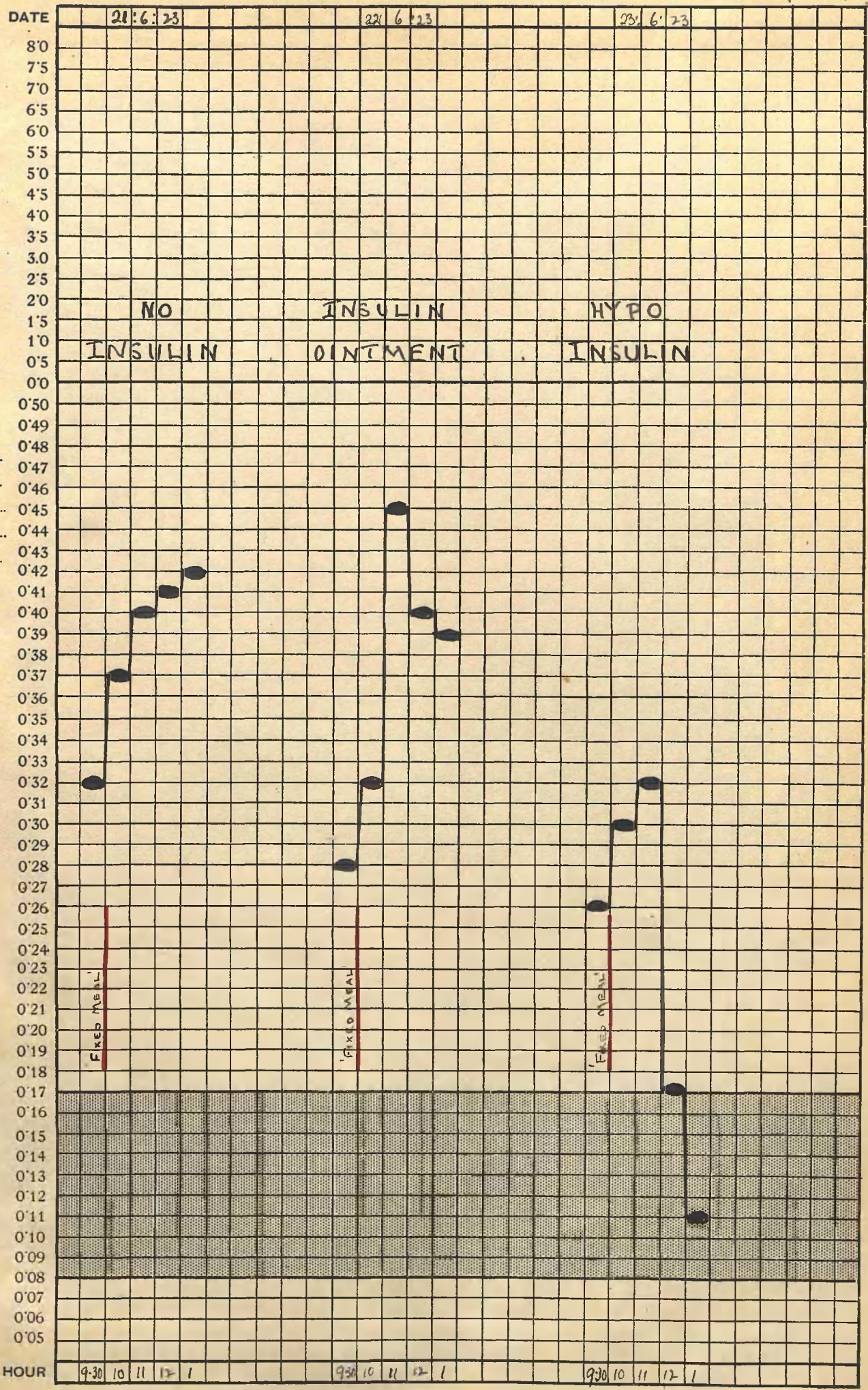
URINARY  
SUGAR.

Gms. 100  
90  
80  
70  
60  
50  
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30  
20  
10  
0





T. 5.



URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission...



## SECTION II

(METHOD OF DISTINGUISHING)

CHARTS.



CHART I

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URINARY  
SUGAR  
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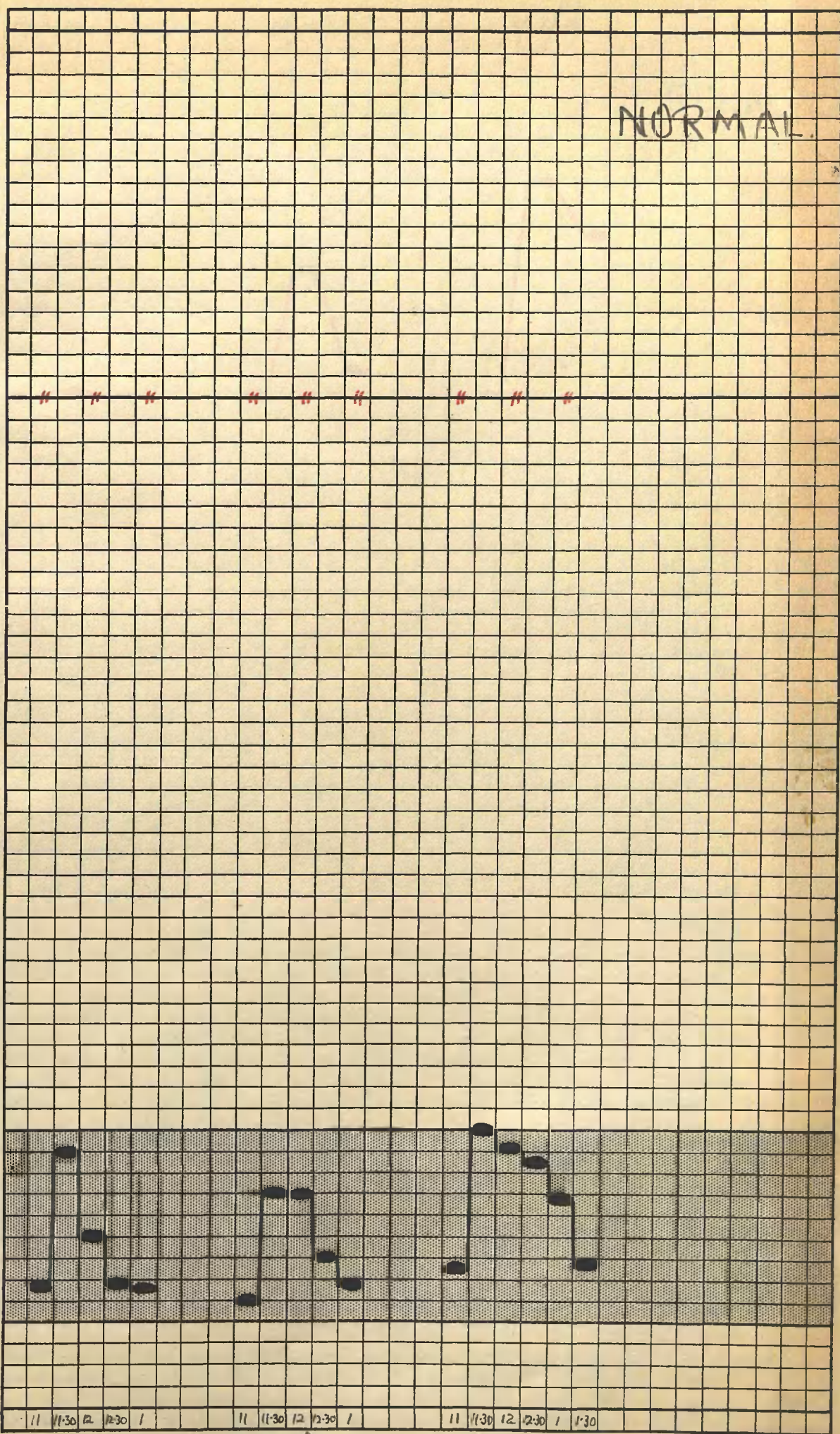
NORMAL

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
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Date of Admission...

HOUR



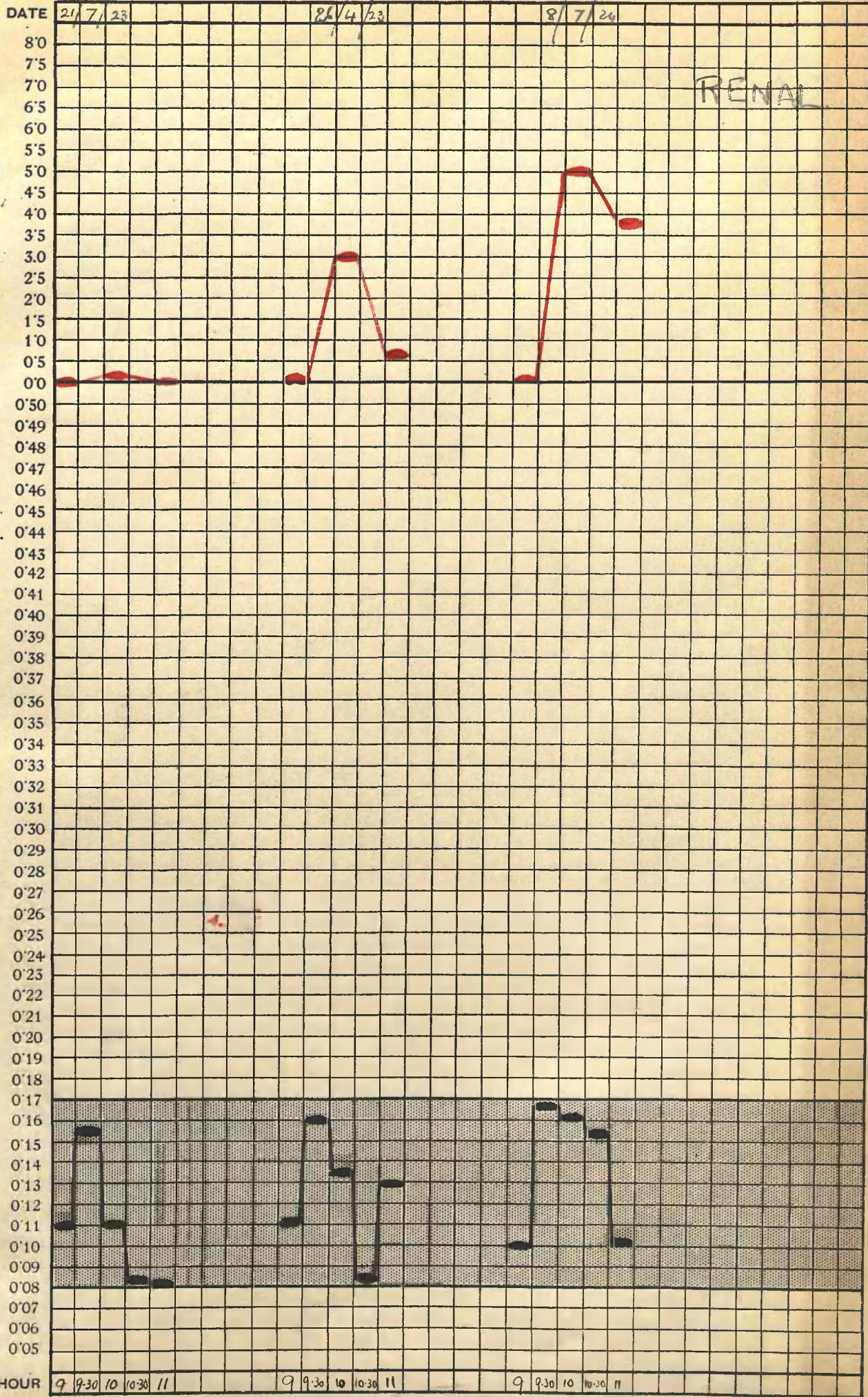
(a)

(b)

(c) (W.R. Age 65)



II



RENAL

URINARY SUGAR %

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD SUGAR %

Date of Admission...

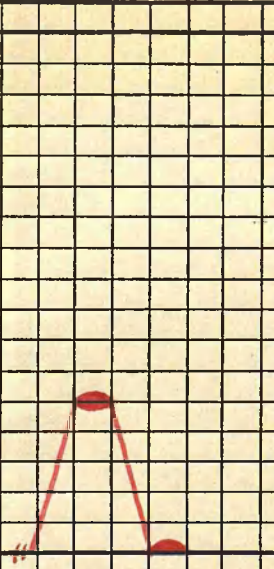
HOUR 9 9:30 10 10:30 11 9 9:30 10 10:30 11 9 9:30 10 10:30 11

1 2 3



LAG

URINARY  
SUGAR  
%



Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%



Date of Admission...

HOUR

9 9:30 10 10:30 11

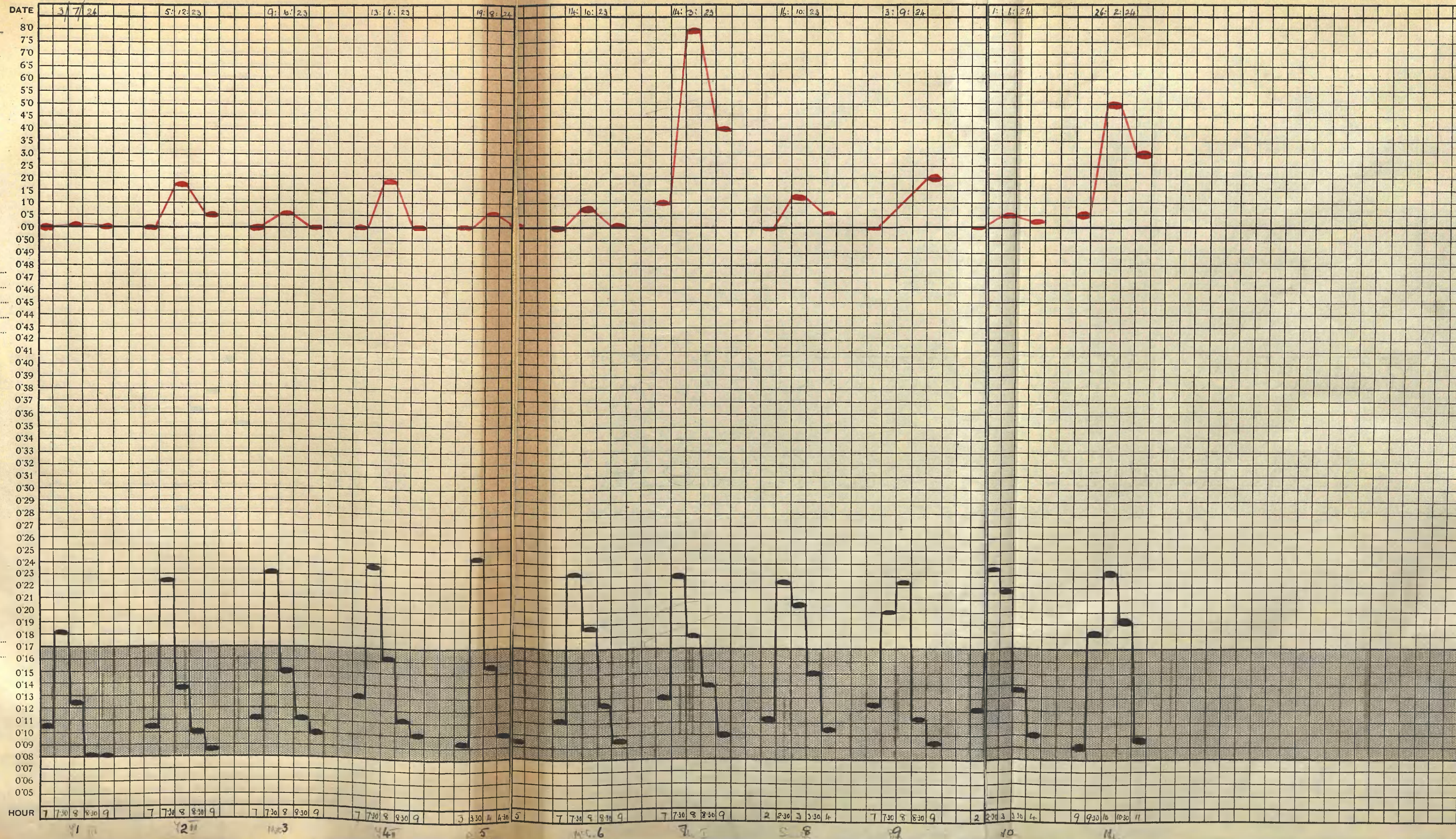


## CHART IV

**URINARY  
SUGAR**  
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BLOOD  
SUGAR  
%

Date of Admission...





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URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission...

HOUR

DIABETIC

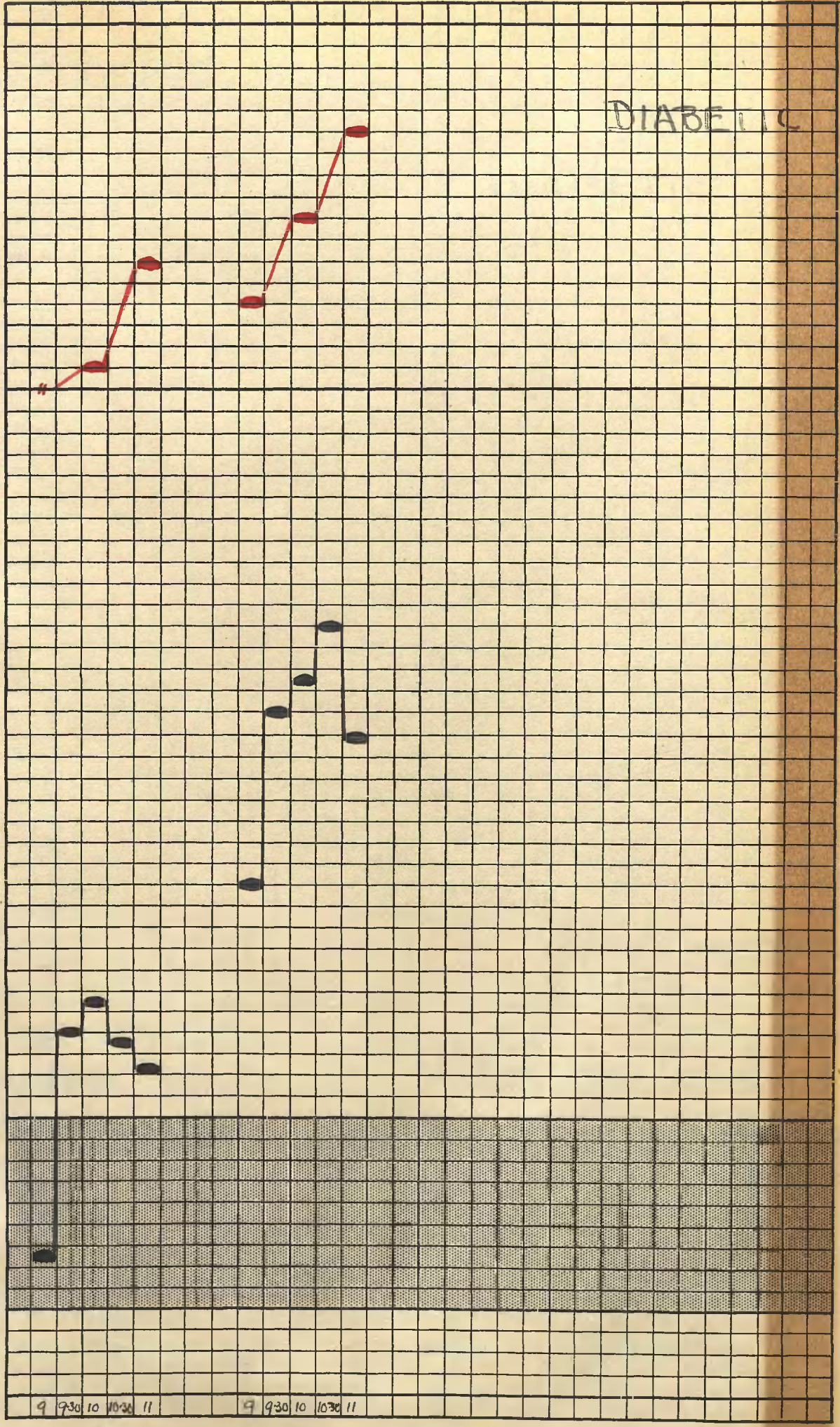




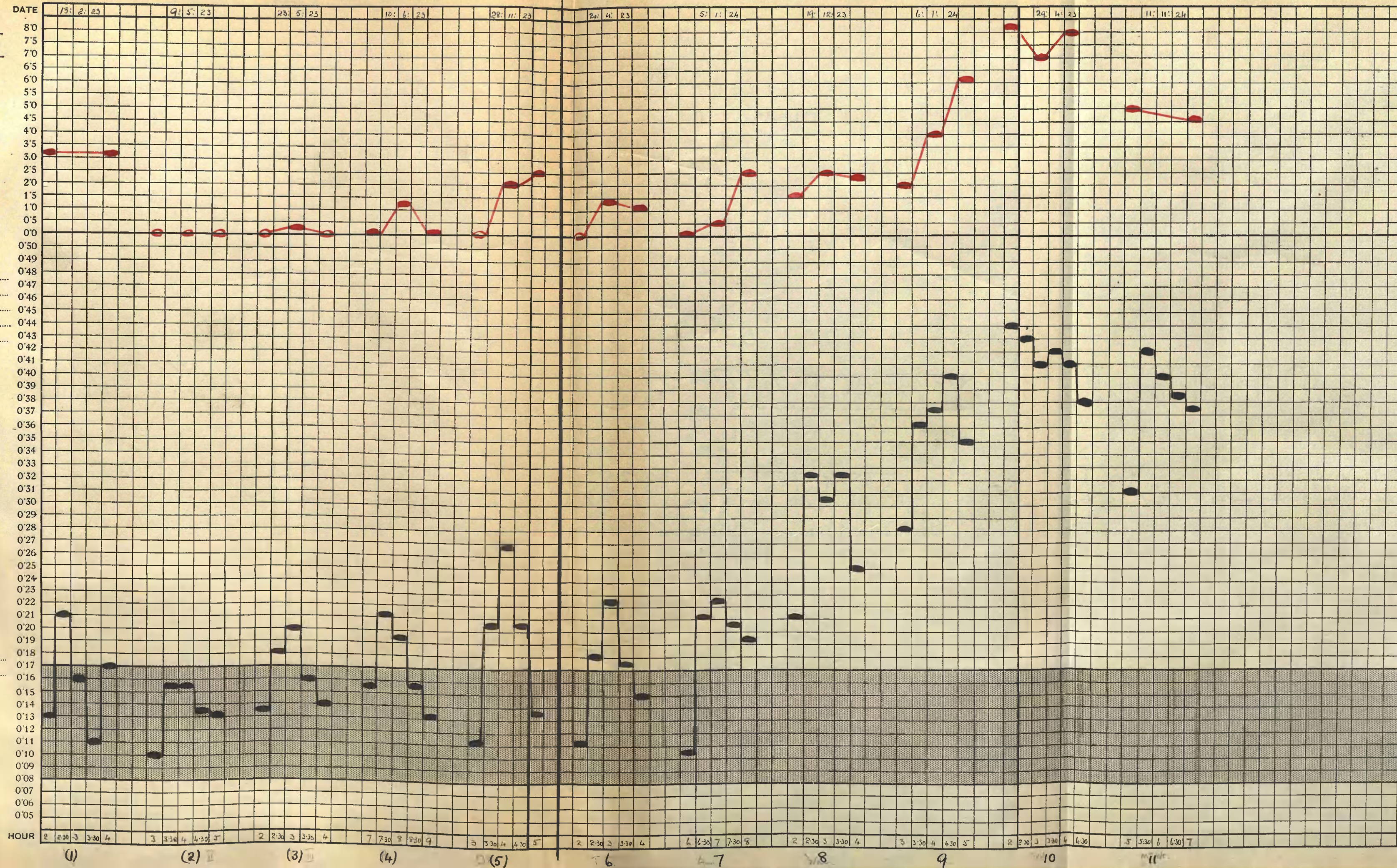
CHART VI

URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

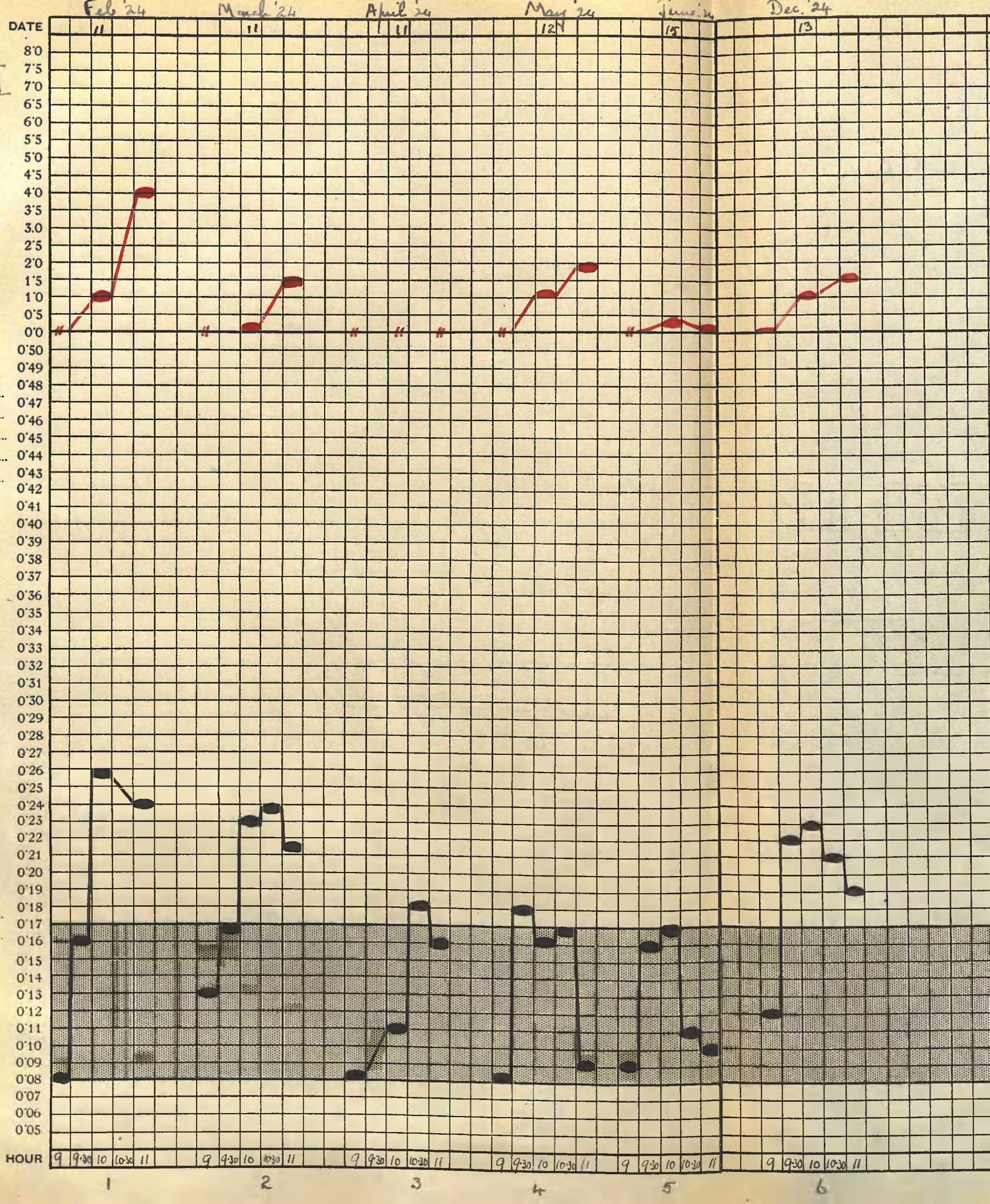
BLOOD  
SUGAR  
%

Date of Admission...





# CHART VII



URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission.....



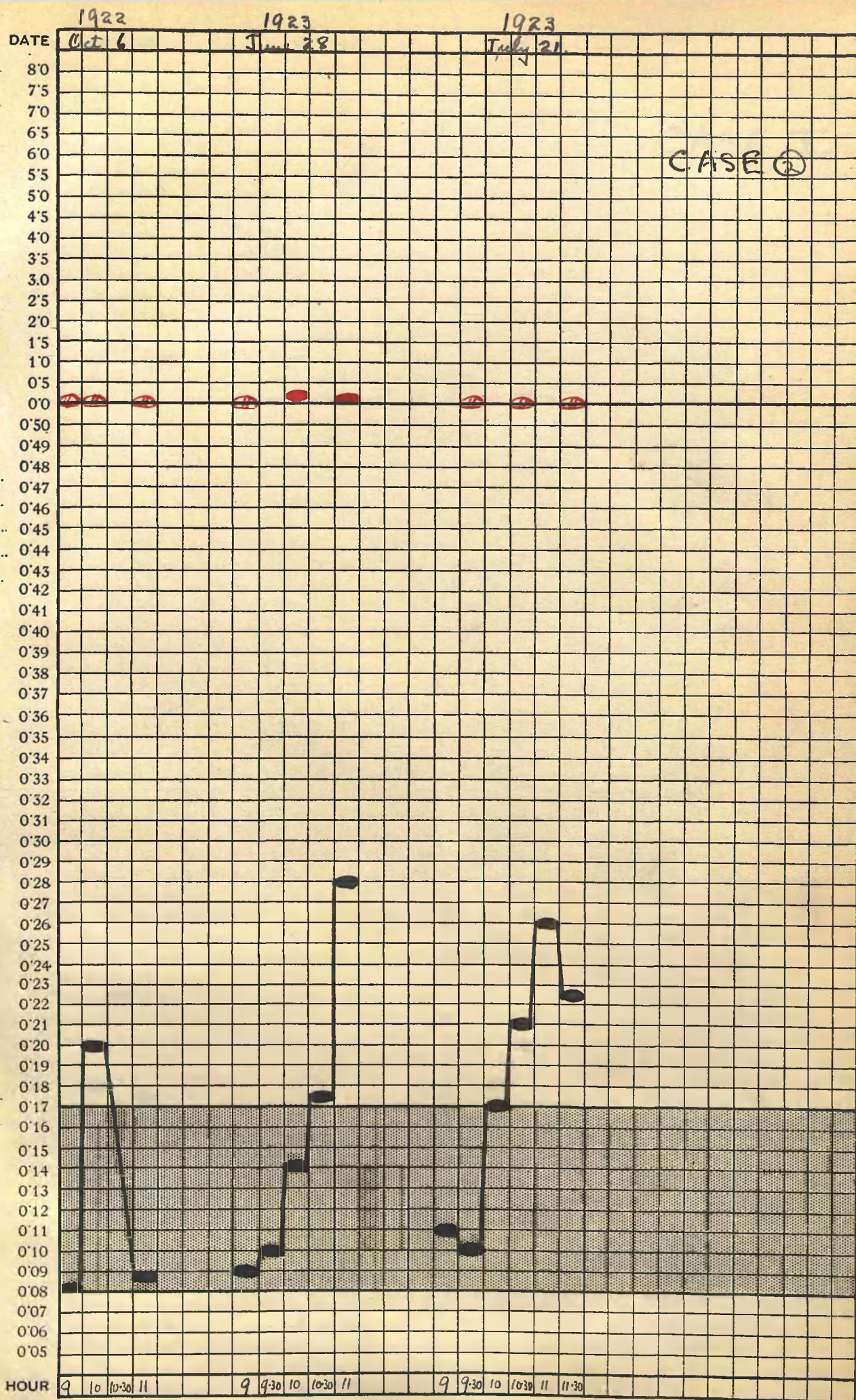
# CHART VIII

URINARY  
SUGAR  
%

Name.....  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission...





DATE

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9: 5: 23

23: 5: 23

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URINARY  
SUGAR  
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Name.....T.

Age.....

Ward.....

Journal.....

Page.....

BLOOD  
SUGAR  
%

Date of Admission...

HOUR

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CASE I



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URINARY  
SUGAR  
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CASE II

Name.....Y

Age.....

Ward.....

Journal.....

Page.....

BLOOD  
SUGAR  
%

Date of Admission...

HOUR

7 7:30 8 8:30 9

7 7:30 8 8:30 9

7 7:30 8 8:30 9

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3



XI

URINARY  
SUGAR  
%

Name.....S  
Age.....  
Ward.....  
Journal.....  
Page.....

BLOOD  
SUGAR  
%

Date of Admission...

